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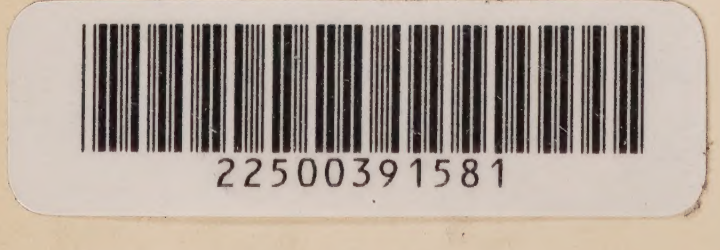
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PREFACE.

THE following articles are presented in bound form to show the character of the original work done by the instructors of the school, or under their personal supervision, during the year. It was not thought desirable to include clinical papers in this collection.

A portion only of the work of the year 1887 is represented by these publications.

J. COLLINS WARREN,
Chairman of the Committee on Publications.

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Extracted from the
American Journal of the Medical Sciences for April, 1887.

THE ACTION OF SULPHURIC ETHER ON THE PERIPHERAL NERVOUS SYSTEM.

FROM EXPERIMENTS PERFORMED IN THE PHYSIOLOGICAL LABORATORY OF THE HARVARD MEDICAL SCHOOL, BY DR. F. H. HOOPER, DR. F. W. ELLIS, AND MR. J. W. PERKINS.

By H. P. Bowditch, M.D.,
PROFESSOR OF PHYSIOLOGY IN HARVARD UNIVERSITY.

In an article on "The Respiratory Function of the Human Larynx" in the *N. Y. Medical Journal* for July 4, 1885, Dr. F. H. Hooper recorded the observation that stimulation of the recurrent laryngeal nerves of dogs causes a dilatation of the glottis when the animals are thoroughly etherized, but a constriction when they are slightly or not at all under the influence of the drug. As the phenomenon occurred equally well whether the stimulation was applied to the intact nerve or to its peripheral end after section it was evident that the action of the drug must be either upon the peripheral portions of the nerve or upon the muscles.

In order to decide between these alternatives, and to ascertain whether similar observations could be made upon other groups of antagonistic muscles supplied by a single nerve trunk, experiments were undertaken by Mr. J. W. Perkins, interne at the Children's Hospital, Boston, in the autumn of 1885.

The sciatic nerve of the frog, with the groups of flexor and extensor muscles which it supplies, was selected for purposes of experiment. The frog was prepared by destroying the medulla and cord. The sciatic plexus was dissected out on both sides and shielded electrodes placed on the nerves close to their origin from the cord. The frog was then suspended in such a way that the hind legs hung freely in fluid contained in two beaker glasses, one leg being immersed in a 0.5 per cent. solution of common salt and the other in a similar solution to which three per cent. of sulphuric ether had been added.

Tetanic stimulation of the sciatic plexus was produced by a Du Bois Reymond induction apparatus having a secondary coil consisting of 10,260 turns of fine wire and graduated with arbitrary units of intensity

such that the maximum stimulation, produced by pushing the secondary completely over the primary coil, corresponded to 1000 units of intensity.

The experiment consisted in applying, at short intervals, a tetanic stimulation of very weak intensity ($I = 0.5 - 2$) to each sciatic plexus in turn and observing the position of the legs as one of them gradually came under the influence of the ether. It was observed that at the beginning of the experiment stimulation produced the same effect on both sides; viz., extension of the leg and abduction of the toes. After the ether had been applied five or ten minutes this effect gave place to a flexion of the leg and adduction of the toes, and after about twenty minutes the stimulation failed to produce any effect. Stimulation of the nerve on the side where the leg was immersed in the salt solution produced the same extension as in the beginning of the experiment. The solutions were now interchanged so that the etherized leg was suspended in the salt solution, and *vice versâ*. As the effect of the ether disappeared from the leg in the salt solution the stimulation of the nerve produced at first flexion and adduction which afterward gave place to extension and abduction as at first. This experiment was repeated with very constant results upon five different frogs.

It thus appears that there is a stage in ether narcosis when stimulation of the nerve, which ordinarily produces extension and abduction, causes the opposite effects and this action can be observed both when the narcosis is coming on and when it is passing off. For the sake of brevity, this action of ether will be termed the "ether effect." The phenomenon is apparently the same as that observed by Hooper on the larynx and, so far as the physiological action of ether is concerned, the constrictors of the larynx seem to correspond to the extensors, and the dilators of the larynx to the flexors of the leg, an analogy the reverse of that which was maintained by Rosenbach,[1] who was led to his conclusions by pathological considerations.

Since in these experiments the central nervous system was destroyed, it is obvious that this "ether effect" must be at the periphery, but whether upon the nerve trunks, the nerve terminations, or the muscular fibres cannot be decided from the above experiments.

To study this question the following experiment was performed. The sciatic nerve was freed from its connections for a distance sufficient to permit the portion of the nerve between the electrodes and the muscles to be immersed in a solution of ether. This was most readily effected by means of a bent glass tube, with a hole blown in the side, such as is represented in Fig. 1. The tube was bent or inclined at such an angle that fluid introduced by means of a pipette into the upper end would flow down and cover a loop of the nerve and could at the same

[1] Bresl. ärztl. Zeitschr., 1880, Nos. 2 and 3.

time be readily washed away and replaced by another fluid introduced in the same way. Five experiments performed in this way gave results which were very constant and quite similar to those obtained when the ether was applied to the skin of the leg. The only important difference was that the action of the ether was much more prompt when applied directly to the nerve, complete paralysis taking place in about seven minutes. The "ether effect" manifested itself both in flexion and adduction and occasionally in peculiar twisting movements of the phalanges due to unequal stimulation of the various muscles. The important observation was also made that whenever flexion was obtained it could be converted into extension *by increasing the intensity* of the stimulation.

Fig. 1.

From these experiments we are justified in concluding that the "ether effect" is a phenomenon dependent upon the action of the drug upon the nerve fibres themselves, though, of course, the possibility of a simultaneous action upon the muscles is not excluded. An attempt was made to ascertain whether any such action of the drug upon the muscles really occurs by poisoning a frog with curare and applying electricity directly to the limb while immersed in a solution of ether.

It was found, however, to be practically impossible to apply the electricity in such a way as to stimulate uniformly all the muscles of the limb and thus produce a movement comparable with that obtained when the muscles are stimulated through the nerve. The experiments made in this way were, therefore, inconclusive. It is evident, however, that the observed action of ether on the nerve trunks is sufficient to explain the "ether effect," and since, when one sufficient cause is demonstrated, it is illogical to assume the existence of another, we may conclude for the present that the phenomenon in question concerns exclusively the nerve fibres.

We must next inquire whether the ether can be supposed to exercise an elective action upon the fibres contained in the nerve trunk and to paralyze the fibres supplying the extensor and abductor muscles before it affects those which go to the flexor and adductor group, or whether some simpler and more probable explanation can be suggested.

The above-mentioned observation that flexion could be converted into extension by increasing the intensity of the stimulation, suggests the idea that the "ether effect" may be simply *partial paralysis* of the nerve

fibres. On this hypothesis we should expect to find that stimulation of the nerve with a *very weak* current would cause flexion of the limb, even without the application of ether.

To test this question and also to study the effect of various other reagents besides ether upon the nerve trunk, a series of experiments on frogs were undertaken in the spring of 1886, by Dr. F. W. Ellis.

In these experiments the frog was prepared in the same way as by Perkins, but the position of the legs resulting from the stimulation of the nerve was recorded by means of photography. For this purpose the legs of the animal were suspended in water contained in a large cell with parallel plate glass sides. Behind the cell a piece of opaque white glass afforded a suitable background and in front was placed a dry plate camera by means of which the position of the legs could be photographed instantaneously. The drugs used were mixed with one-half per cent. solution of sodium chloride in distilled water and applied to the nerve in the manner above described.

The ordinary effect of an electrical stimulation of the sciatic nerve is well shown in Fig. 2.[1] Even with quite weak currents there is ordinarily marked extension of the knee, ankle, and toes, accompanied by strong abduction of the toes. In other words, the position of the leg is that assumed when the animal is just completing a vigorous stroke in swimming.

Fig. 2.

Effects of Ether.—In the experiments with ether, solutions of the drug varying in strength from one-half to five per cent., were employed. A three per cent. solution was most frequently used and gave, upon the whole, the best results. The observations of Perkins were in the main confirmed—*i. e.*, there was found to be a stage in the paralyzing action of the drug when stimulation of the nerve caused the leg to assume a position contrary to that occasioned by the same degree of simulation without ether. Instead of extension at the knee and ankle, there might be flexion; instead of extension and abduction of the toes, there might be flexion or adduction, or both. It was rare that all these contrary effects were manifested at once. There was generally a predominance of one, and frequently only one was manifested. These "ether effects" were obtained with some frogs more easily than with others, and were not always constant for the same frog. They were generally better obtained when

[1] This and the following figures are photoelectrotype reproductions of photographs taken in the manner above described. As the frogs were suspended with their ventral surface toward the camera the right leg of the animal appears on the left of the figure, and *vice versâ*.

the nerve was recovering from the effects of ether (*i. e.*, after removal of the ether solution from the tube and washing the nerve with saline solution) than when it was coming under the influence of the drug.

Fig. 3.

Ether on nerve.

Fig. 4.

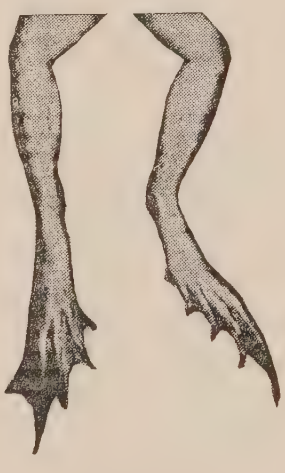
Ether removed.

Figs. 3–8 illustrate these "ether effects." Fig. 3 shows the position assumed by the left leg on irritation of the sciatic nerve during the local application of ether. Fig. 4, the position assumed by the same leg after the removal of the ether. Here flexion and adduction under ether gave place to extension and abduction on removal of the drug. Figs. 5 and 6 show a similar "ether effect" on the right leg, but confined in this case to the muscles moving the toes.

Fig. 5.

Ether on nerve.

Fig. 6.



Ether removed.

In Figs. 7 and 8 the "ether effect" manifests itself in a flexion of the foot and adduction of the toes combined with a strong extension of the leg.

The local application of ether to the nerve was found not to affect the irritability of the nerve below the point of application. This was shown by experiments in which electrodes were placed upon the nerve, both above and below the ether tube. When powerful stimulation applied above the tube no longer caused contraction of the muscles,

a very slight current applied below would produce the usual effect. The ether, therefore, seemed to produce a change in the nerve at the point of application, which rendered it incapable of transmitting the

Fig. 7.

Ether on nerve.

Fig. 8

Ether removed.

ordinary nerve impulse, complete paralysis resulting when the action of the drug was pushed to its extreme, and the so-called "ether effects" during intermediate stages.

Experiments with chloroform and alcohol showed that the "ether effects" could be obtained with these drugs also, although not so strikingly as with ether. A full discussion of the action of these and other drugs will be given on a future occasion.

It was also found, as had been previously observed by Perkins, that an increase in the intensity of the stimulation caused the "ether effect" to disappear, and the ordinary effect of nerve irritation (viz., extension and abduction) to take its place. This led to experiments on the effect of variation in the intensity of the stimulus, and particularly on the

Effects of very Weak Currents.—The first experiments showing the effect of very weak stimulation were accidental. It was noticed occasionally, when the right sciatic plexus was very strongly stimulated in order to ascertain whether the application of ether to the nerve of that side had produced complete paralysis, that movements of flexion were produced in the leg of the opposite side. These were obviously due to the escape of electricity upon the left plexus, and it was observed that a still further increase in the intensity of the stimulus applied to the right plexus caused the ordinary movements of extension and abduction on the left side. It was subsequently found that by employing weak currents it was generally possible to cause movements (viz., flexion and adduction), which were ordinarily excited by very much stronger stimuli after the application of ether to the nerve. To produce these effects, it was necessary to use currents of very feeble intensity. These were best obtained by separating the secondary coil of the induction apparatus forty or fifty centimetres from the primary, and then turning it round a vertical axis. When flexion and adduction had been obtained in this

way it was always possible to produce the ordinary movements of extension and abduction by increasing the intensity of the stimulation. Occasionally, as a result of very weak stimulation, twitchings or clonic contractions of the muscles were produced. These seemed to be due to a conflict between opposing sets of muscles.

Fig. 9.

Weak irritation.

Fig. 10.

Strong irritation.

Figs. 9–13 illustrate the effects of variations in the intensity of the stimulation applied to unetherized nerves. Fig. 9 shows the position assumed by the left leg under the influence of a very feeble stimulus applied to the sciatic nerve of that side, and Fig. 10 the effect of a stronger stimulus applied to the same nerve.

Fig. 11.

Weak irritation.

Fig. 12.

Strong irritation.

Figs. 11 and 12 show, in a similar manner, the effect of increasing the intensity of the stimulus applied to the right sciatic nerve. Fig. 13 shows the effect of a weak irritation of the left sciatic nerve, and by comparing the position of the left leg in this figure with that of the right leg in Fig. 2, a good idea can be obtained of the great variety of movement which can be produced by changes in the intensity of the stimulation.

These experiments by Ellis prove beyond a doubt that the effects observed by Perkins on stimulating a nerve immersed in a solution of ether, can be obtained with other drugs, and often with very weak currents without any drug. Without ether, flexion and adduction are to be obtained only with very feeble currents, but with ether much stronger stimulation produces the same effect. In both cases, however, an increase in the intensity of the stimulus causes these movements to give place to extension and abduction; the effect of the ether may, therefore, be said to consist in transferring the point, on the scale of intensity, at which the effect of nerve irritation changes from flexion and adduction to extension and abduction from the region of weak to that of relatively strong stimulation.

Fig 13.

Weak irritation.

The "ether effect," as observed in the sciatic nerve of the frog, may be best explained by supposing that a partial paralysis of the nerve by the drug converts what would naturally be a strong into a weak irritation, and that this weak irritation affects only the flexor group of muscles because these are, for some reason or other, more irritable than their antagonists. A stronger irritation or a removal of the ether causes extension, since when both flexors and extensors are stimulated, the position of the leg is determined by the latter—*i. e.*, the more powerful group.

The question of the difference in irritability of the flexors and extensors of the frog's leg and the cause thereof has been much discussed. The fact that a feeble irritation of the sciatic causes flexion, and a strong irritation extension of the leg was first observed by Ritter in 1798, but the subject was first thoroughly investigated by Rollett in 1874–75.[1] This observer experimented with the amputated leg suspended in the air, and also with the muscles attached to myographic levers, and reached the conclusion that the cause of the greater irritability of the flexors was to be sought in the nerve fibres or in their mode of attachment to the muscles. Bour,[2] also, while criticising some of Rollett's methods, found that when the irritation was applied to the upper portion of the sciatic nerve, the flexors were called into action by a feebler stimulus than the extensors.[3]

1 Sitzungsberichte der Wiener Acad. Dritte Abth., lxx. S. 7; lxxi. S. 33; lxxii. S. 349.

2 Verhandl. d. Phys. med. Gesells. in Würzburg. N. F. viii. 221.

3 Dr. W. P. Lombard has kindly made a few experiments upon the irritability of the muscles moving the knee-joint of the frog, using for the purpose the delicate apparatus employed by him in studying the reflex actions of the frog (Archiv für Physiologie, 1885, p. 408). These experiments, though interesting in themselves, are not conclusive as to the relative irritability of the flexor and extensor groups of muscles. The evidence, however, seems to be slightly in favor of the above mentioned conclusion of Bour.

There seems, therefore, to be no doubt that the flexor apparatus of the frog's leg responds to a feebler simulation than the extensor, but whether the cause of this difference lies in the nerve fibres, the muscle fibres, or in the mode of connection between the two, must be left for future investigations to determine. It is not impossible that the microscope may reveal histological differences between the different sorts of muscles similar to those described by Ranvier and others between the red and white muscles of the rabbit.

We must now inquire whether the above explanation of the "ether effect," as studied on the sciatic nerve of the frog, is applicable to the action of the drug, as observed by Hooper, on the recurrent laryngeal nerve of the dog.

A single observation upon a chloralized dog is reported by Hooper, in which a thread passed through the nerve and left in position for several days produced an effect analogous to that of ether on the sciatic nerve of the frog, viz., abduction of the vocal cord on irritation of the nerve on the side thus operated upon, while the same irritation on the opposite side produced the ordinary effect, viz., adduction. As all attempts to repeat this observation failed, no importance can be attached to it. On the other hand, it had never been observed in Hooper's experiments that feeble irritations caused a dilatation of the glottis, while stronger ones caused a constriction, but, as the effect of *very feeble* stimuli had not been specially studied, it was thought desirable, in view of the positive results obtained by Ellis on the frog, to make a few additional experiments directed to this particular point. Experiments were accordingly made upon chloralized dogs, with the result of showing that feeble irritations differed from strong ones in the extent but not in the character of the movements produced. Nothing resembling a dilatation of the glottis could be produced by diminishing the intensity of the stimulus.

When this point had been reached in the study of the question, a paper entitled "The Function of the Recurrent Laryngeal Nerve," by F. Donaldson, Jr., B.A., M.D., appeared in *The American Journal of the Medical Sciences* for July, 1886. In this paper the writer gave an account of experiments on dogs similar to those made by Hooper, but leading to different results, for Donaldson, though unable to obtain the above described "ether effect," did observe a dilatation of the glottis when the recurrent nerve was irritated with *very weak* stimuli. This observation is, therefore, precisely in harmony with Ellis's results obtained upon frogs, and if confirmed would lead to the conclusion that the "ether effect" is in these cases also an effect of partial paralysis.

Shortly after the appearance of Donaldson's article a paper was read at the meeting of the British Medical Association in Brighton, entitled "On an Apparently Peripheral Differential Action of Ether upon the

Laryngeal Muscles," by Felix Semon, M.D., F.R.C.P., and Victor Horsley, B.S., F.R.S. The authors were able to confirm the results of Hooper's experiments as to the effect of ether on the recurrent laryngeal nerve, and even gave a graphic representation of the "ether effect" studied by means of a rubber bulb inserted between the vocal cords. They explained Donaldson's failure to obtain the "ether effect" on the supposition that the animal was not sufficiently etherized. They also repeated Donaldson's experiments with currents of varying strength and obtained similar results in those cases in which the animal was not deeply under the influence of ether.

In view of these somewhat discordant results a renewed investigation of the effect of electrical irritation of the recurrent laryngeal nerve, with and without narcotics, seemed desirable, and Dr. Hooper is now engaged in a series of experiments which it is hoped will shed further light upon the subject.

The detailed results of these experiments will be published on some future occasion, but at the present time the following preliminary statement may be made.

It has not been found possible in these experiments to obtain a dilatation of the glottis by any stimulation of the recurrent laryngeal nerve unless the animal is under ether.

When the animal is etherized, the effect of irritating the nerve varies with the depth of the narcosis and the strength of the irritation.

If the animal is thoroughly under the influence of the drug dilatation of the glottis is produced by irritations of all intensities. In less profound etherization dilatation is still obtained with feeble irritation, but gives place to constriction when a stronger stimulus is applied.[1] As the effect of the ether passes off constriction occurs with feebler and feebler intensities until, finally, dilatation can no longer be obtained, and constriction is produced by irritation of all intensities.

The observation of Donaldson that dilatation is produced by feeble and constriction by strong stimuli is, therefore, correct *for a certain stage of etherization*, but for *unetherized* animals his statement has not been confirmed. The cases in which Donaldson has recorded a dilatation as a result of feeble stimulation are those in which the animal had been previously etherized, but was supposed to have recovered from the influence of the drug. In view of Hooper's failure to obtain dilatation on unetherized dogs, it seems probable that in Donaldson's cases the drug had not been completely eliminated.

It will thus be seen that, while the "ether effect," as shown on the sciatic nerve of the frog, may be explained as a partial paralysis of the nerve by the drug, the same explanation cannot be given of the phe-

[1] It will be observed that this result is in harmony with the above-mentioned observations of Semon and Horsley.

nomenon as manifested on the recurrent laryngeal nerve of the dog, for the action of ether cannot be imitated by feeble irritation of the nerves of unetherized animals.

The local action of ether, applied directly to the nerve, presents another point in which the two phenomena differ from each other, for the distinct "ether effects" obtained in this way by Perkins and Ellis on the frog's sciatic do not manifest themselves on the laryngeal nerve of the dog. A six per cent. solution of ether dropped upon the nerve of chloralized dogs, between the electrodes and the larynx, was found to gradually paralyze the nerve, so that stronger and stronger stimuli were needed to constrict the glottis, but a dilatation was, under these circumstances, never observed.

Conclusions.—The principal results of these various researches may be summarized as follows:

1. The ordinary effect of electrical stimulation of the recurrent laryngeal nerves of dogs is to cause constriction of the glottis, but if the animal is thoroughly etherized dilatation may be produced.

2. If the animal is partially etherized, the effect of the stimulation will vary with the strength of the current, a weak irritation causing dilatation, and a stronger irritation causing constriction of the glottis.

3. The more complete the etherization the greater is the intensity of the current necessary to produce constriction.

4. A similar "ether effect" may be observed when the sciatic nerve of the frog is stimulated, the ordinary extension of the leg and abduction of the toes giving place, under the influence of this drug, to flexion and adduction. It is, however, always possible to produce the ordinary effects by increasing the intensity of the stimulation; and, on the other hand, it is possible, by using very feeble stimuli, to produce without etherization the same effects which occur on the application of stronger stimuli to animals under the influence of the drug.

5. This "ether effect" on frogs may be observed both when the ether solution is introduced by cutaneous absorption, and when it is applied locally to the nerve below the point of stimulation. Hence the most reasonable explanation of the phenomenon is that the drug, by diminishing the conducting power of the nerve, causes a strong irritation applied to the nerve to become a weak irritation when it reaches the muscles.

6. This explanation cannot be applied to the phenomenon as exhibited by the recurrent laryngeal nerve of the dog, for without ether feeble irritations do not produce dilatation, and, with complete etherization, strong stimuli do not produce constriction of the glottis.

Reprinted from the New York Medical Journal for July 9, 16, 23, and August 6, 1887.

THE ANATOMY AND PHYSIOLOGY OF THE RECURRENT LARYNGEAL NERVES.*

By FRANKLIN H. HOOPER, M. D., Boston,
FROM THE PHYSIOLOGICAL LABORATORY OF THE HARVARD MEDICAL SCHOOL.

I. Anatomy.

NOTWITHSTANDING the centuries that have elapsed since the reputed discovery of the recurrent laryngeal nerves by Galen, it is doubtful if there be another pair of nerves in the body which have given rise to so many diverse and fanciful theories to account for the anatomical peculiarity of their course or to elucidate their physiological functions. At the present day our knowledge of the anatomy of these nerves is, for the most part, complete and exact. The reason why they are not given off by the pneumogastrics at a point opposite the larynx so as to reach that organ by the shortest route, instead of branching from the pneumogastrics low down in the neck and running a complicated and recurrent course to the larynx, is now clearly understood. Yet Magendie,† in his lectures on the nervous system, delivered at the Collège de France at so recent a date as 1836, asserted that this was a problem which anatomists were unable to solve. But several years prior to the date of Magendie's lectures two articles had appeared in a Scotch journal which, had they come under his notice, would have given him a clew to the explanation of the matter.

Stedman, in 1823, in a paper entitled "A Singular Distribution of some of the Nerves and Arteries in the Neck and the Top of the Thorax,"‡ gives a description and a drawing of his dissection

* Read before the American Laryngological Association at its ninth annual congress.

† "Lancet," July 1, 1837, p. 503.

‡ "Edin. Med. and Surg. Jour.," xix, 1823, p. 564.

of an aged female in whom he found that the right recurrent laryngeal nerve was absent. There were, however, nerves given off from the trunk of the pneumogastric, about the middle of the neck, which went direct to the larynx. He also found, in the same subject, an irregularity in the origin and course of the right subclavian artery. It arose from the arch of the aorta behind and a little to the left side of the left subclavian artery, and, forming an arch, pierced between the œsophagus and vertebral column in the region of the first vertebra of the back, and then passed over the first rib on the right side. Stedman was the first to cite an example of the connection between an irregular origin of the right subclavian artery and right recurrent laryngeal nerve—a connection which we now know is constant; but he dismissed the subject with the comment that the nerve, in his case, which went direct to the larynx from the pneumogastric probably performed the same functions as the recurrent, and that nature seemed to have been forced to have recourse to this arrangement from the singular situation of the subclavian. Three years after the appearance of Stedman's paper, Hart (1826) reported in the same journal* "A Case of Irregular Origin and Course of the Right Subclavian Artery and Right Inferior Laryngeal Nerve." The artery arose from the aorta direct, and the nerve, instead of being recurrent, went straight from the pneumogastric to the larynx. Hart speculates upon the reason why the nerve in this instance should have been straight and short, and, although his views are not strictly in accord with the teaching of modern embryologists, they show that he was aware that the secret of the circuitous course of the inferior laryngeal nerves was to be sought in the natural law of embryonic development. He says: "In the earlier periods of the existence of the fœtus the rudiment of the head appears as a small projection from the upper and anterior part of the trunk, the neck not being yet developed. The larynx at this time is placed behind the ascending portion of the arch of the aorta, while the brain, as it then exists, is situated so low as to rest on the thymus gland and front of that vessel. Hence it is that the inferior laryngeal nerves pass back to the larynx, separated by the ascending aorta, the left going round its arch, while the right goes below the *arteria innominata*. As gestation advances, the head becomes more distinct, and the neck begins to be formed after the second month, which, as it lengthens, has the effect of moving the brain upward to a greater distance, and of drawing out the larynx from the chest, in accommodation to which

* "Edin. Med. and Surg. Jour.," April, 1826, p. 286.

the nerves of the par vagum and their recurrents become elongated, and hence the circuitous route the latter are found to take afterward, forming loops in which the aorta and right subclavian artery are, as it were, suspended. . . . Had not the great blood-vessels been originally thus interposed between the brain and larynx, the inferior laryngeal nerves would not have been entangled by them, and we should find them in the adult taking the nearest route to their destination."

These early papers upon this subject are most interesting, and show that their authors were careful observers. They pointed out the right path, which, however, was not followed by all subsequent writers who expressed themselves on the question.

Swan, for instance, entertained certain theories in regard to the influence which the blood-vessels had upon the nervous system, and in "An Essay on the Connection between the Action of the Heart and Arteries and the Functions of the Nervous System," London, 1829, p. 50, he applied his views in a very curious way to explain the recurrent course of the inferior laryngeal nerves. After speaking of their encompassing the subclavian artery on the right side and the arch of the aorta on the left, and remarking that it was very singular that neither of the carotids was included, he says: "I conceive this disposition was intended for producing a more extensive sympathy between the arteries of those parts of the body which are subjected to be excited by exercise, and the glottis; for when the action and distension of the arteries are increased by exercise, the recurrent nerves become stimulated, and consequently the muscles of the glottis, the opening of which thus becomes widened, so as to admit a greater and more free supply of air into the lungs. It may be supposed that any other disposition would have sufficed, so long as the parts were properly supplied with nerves, and a case is related by Dr. Stedman where the recurrent was wanting on the right side, and its place was supplied by numerous branches of the par vagum; but in this instance 'the right subclavian artery rose from the arch of the aorta behind and a little to the left side of the left subclavian, and, forming an arch, pierced between the œsophagus and vertebral column in the region of the first vertebra of the back, and then passed over the first rib on the right side.' It must be remarked that the right subclavian was already under the influence of the left recurrent, and therefore the usual distribution was not necessary on the right side. The same unusual distribution I have also observed in a rabbit, which was very healthy, but I never saw it in exercise,

and therefore can not determine whether its respiration was affected during that state. It is a curious circumstance that the recurrents do not always include the carotids; but it seldom happens that the action of these arteries is much increased, except by the exertions of the voice, as in singing, etc., and therefore, if they had been included, the delicate and complicated actions of the glottis, which are produced during these processes, might have been too much interfered with, and I believe a compensation is made for this omission by the connection of the pharyngeal plexus with the superficial cardiac nerve. . . . The subclavian artery on the right side and the aorta on the left are almost encircled by the par vagum and its recurrent branches. This distribution, no doubt, connects the actions of these arteries with those of the glottis."

Hilton (1837), to whom we are indebted for an admirable paper * describing the distribution of the recurrent nerves, states in a footnote that he had seen a subject in his dissecting-room in which the right subclavian artery came off from the posterior part and left side of the transverse portion of the aorta. In this case, he continues, "the right recurrent nerve did not curve round the artery, but was detached from the pneumogastric at an acute angle with the descending part of the nerve, about opposite the fifth cervical vertebra." We find, moreover, in the same writer's well-known "Lectures on Rest and Pain," † the statement that he had seen examples in which the recurrent did not wind around the arch of the aorta or the subclavian artery, yet the course of the nerve was, notwithstanding, equally recurrent, thus clearly indicating that it had no necessary relation with the subclavian artery or the aorta. He then sets forth the following singular theory to explain the course of these nerves: "The nervous influence, whatever it may be, which travels by these recurrent nerves, goes from below upward. And I think it will be apparent why this nerve takes its course from below upward. It is an essential thing, to my mind, that the muscles which are acting upon the air as it escapes outward from the lungs so as to make the voice, should be acting from within outward—that is, from the lower part of the larynx to the upper. It is quite obvious that if they acted in the other way we should all be ventriloquists, talking inwardly to ourselves, as it were, and having no external voice; and it is for the purpose of determining the direction of influence from within out-

* "Guy's Hosp. Reports," vol. ii, 1837, p. 514.

† Second edition, London, 1877, p. 217.

ward that we find this nerve pursuing this singularly recurrent course."

Meckel * says the recurrent nerve is sometimes double, and, though rare, when this occurs it is always on the right side. He considers it probable "that the recurrent nerve results from the plexiform division of the trunk of the pneumogastric, and that its existence is connected with the primitive shortness of the neck, since the larynx is much nearer its origin in the early periods of life than subsequently. This hypothesis would explain its arrangement in the same manner as the high origin and long course of the spermatic vessels."

Daremberg, † in speaking of the recurrent nerve twisting round the subclavian artery on the right side and the arch of the aorta on the left, says: "Que la nature a disposeés comme deux bornes qui les fixent dans la carrière qu'ils ont à fournir avant de se répandre dans les muscles abaisseurs du larynx, qu'ils font mouvoir comme avec une main."

If we wish to obtain a clear idea of the reason why the recurrent nerves recur, we must leave this speculative ground and begin at the beginning of the development of the embryo. These remarkable nerves exist at a very early period of fœtal life. At a certain stage of embryonic development they have, indeed, a transverse direction, and proceed direct from the pneumogastrics to their destination, and their recurrent course is determined later solely through developmental changes in the branchial apparatus of the embryo, and to the descent of the heart into the thorax. We will briefly review these changes: In the cervical region are developed the five pairs of aortic arches—a transitory fœtal apparatus in the higher vertebrates. Of the five pairs of arches, but two are permanent at birth, namely, the fourth arch on the left side, which is represented by the arch of the aorta, and the corresponding arch on the right side, which has become the right subclavian artery. The recurrent nerves originally passed under the fifth pair of aortic arches; but as the *ductus arteriosus*—the fifth arch on the left side—is obliterated after birth, it follows that the left recurrent nerve is hooked under the first permanent arch above it, namely, the fourth, which has become the arch of the aorta; while on the right side the fifth aortic arch disappears entirely, and the right recurrent consequently passes under the fourth aortic arch, which persists as the right subclavian artery.

* "Manual of Anatomy." Translated by Doane, 1832.

† "Exposition des connaissances de Galien," etc., Thèse, Paris, 1841, p. 57.

The long course of the recurrents, like the elongation of the carotids and other anatomical features of the cervical region, is brought about by the change of position which the heart and great vessels undergo during embryonic life, and which we may best describe by borrowing Huxley's words:* "At first the heart of a mammal lies under the middle of the head, immediately behind the first branchial arches, in which the first pair of aortic arches ascend. As the other pairs of aortic arches are developed the heart moves backward, but the fourth pair of branchial arches, by the modification of one of which the persistent aorta is formed, lies, at first, no farther back than the occipital region of the skull, to which the fourth pair of branchial arches belongs. As the two pairs of cornua of the hyoid belong to the second and the third branchial arches, the larynx is probably developed within the region of the fourth and fifth branchial arches; hence the branches of the pneumogastric, with which it is supplied, must, originally, pass directly to their destination. But, as development proceeds, the aortic arches and the heart become altogether detached from the branchial arches and move back, until at length they are lodged deep in the thorax. Hence the elongation of the carotid arteries; hence also, as the larynx remains relatively stationary, the singular course in the adult of that branch of the pneumogastric, the recurrent laryngeal, which primitively passed to the laryngeal region behind the fourth branchial arch, and consequently becomes drawn out into a long loop, the middle of it being, as it were, pulled back by the retrogression of the aortic arch into the thorax."

The proof that the course of the recurrent nerves is a question of development is found in the fact that when, from any cause operating in early fœtal life, irregularities of the arch of the aorta or in the origin of its primary branches exist, the recurrent nerves have always in such instances an anomalous origin and course. There are several recorded cases of this inter-relation of these anomalies. We have already mentioned Stedman's paper, in which he described, for the first time, a case of irregular origin of the right subclavian artery associated with absence of the right recurrent nerve. In this case, undoubtedly, the fourth right aortic arch disappeared very early, and, the right subclavian arising from the descending portion of the aorta, there was nothing to carry the right recurrent down, and it consequently ran directly to the larynx. Through the kindness of Pro-

* "A Manual of the Anatomy of Vertebrated Animals," London, 1871. We take the liberty of substituting the word "branchial" for "visceral."

fessor Dwight we have had an opportunity of observing a similar case in the dissecting-room of the Harvard Medical School. Hérard* in 1846 showed a specimen at the Anatomical Society of Paris, in which the right recurrent was given off from the pneumogastric opposite the cricoid cartilage, and he suggested that we ought to be prudent in denying the existence of the recurrent if we did not find it in its proper place. The left recurrent was normal. He makes no mention of any anomaly of the blood-vessels. Reid,† in 1847, gives the correct explanation of the origin of these anomalies, and says that in those cases of monstrosity where the head and larynx are double, and the two bodies are fused together immediately below this, so that the lower part of the neck, the thorax, and thoracic extremities are single, and where consequently we have four vagi nerves in the upper part of the neck and only two at the lower part, the right recurrent of the right larynx hooks round the subclavian artery, and the left recurrent of the left larynx hooks round the arch of the aorta, while the left recurrent of the right larynx and the right recurrent of the left proceed to their destination from the pneumogastrics as they pass the larynges. Demarquay‡ (1848) was, we believe, the first one in France to record a case of irregular origin of the right subclavian artery with absence of the recurrent nerve on the same side.

Professor Turner, in his admirable paper,# cites cases where there was no innominate artery and an unusual origin of the right subclavian. In one instance the recurrent turned round the inferior thyreoid artery, and in others it passed directly inward to the larynx. He also describes cases where the aorta arched to the right side, in which the left recurrents wound round the obliterated ducti arteriosi while the right recurrents passed behind the arches of the aorta. Krause and Telgmann‖ add to the number of irregular origins of the subclavian artery and recurrent nerves, which, according to Turner, occur once in two hundred and fifty cases.

More recently, Brenner,△ in an interesting paper, describes two instances of this anomaly, and a third where the right recurrent

* "Bull. de la Soc. anat. de Paris," 1846, p. 111.

† Todd's "Cyclopædia," article "Par Vagum," 1847.

‡ "Gaz. méd. de Paris," 1848, p. 714.

"On Irregularities of the Pulmonary Artery, Arch of the Aorta, etc.," "Brit. and Foreign Med.-chirurg. Review," vol. xxx, 1862, p. 173.

‖ "Die Nervenvarietäten des Menschen," Leipsic, 1868.

△ "Ueber das Verhältniss des Nervus laryngeus inferior vagi u. s. w." "Arch. für Anat. und Entwickelungsgesch.," 1883, p. 373.

branched from the pneumogastric opposite the sixth cervical vertebra, and turned round the vertebral artery to reascend to the larynx. Here the right vertebral artery represented the fourth right aortic arch. The branches from the aorta in this case were in this order: First, a vessel that divided some two inches from its origin into the right vertebral and the right carotid; then came the left carotid, the left vertebral, the left subclavian, and finally the right subclavian from the descending aorta. He further reports two cases where the aorta passed over the right bronchus, in which the left recurrents passed under the fifth left branchial arches represented by the obliterated ducti arteriosi. The last writer who has called our attention to this subject is Chaput, whose "Note sur un rapport peu connu du recurrent gauche" may be found in the records of the "Soc. anatom. de Paris," July, 1884.

To return now to the normal relation of the recurrent nerves to the large blood-vessels, we find (Luschka) that the left recurrent leaves the pneumogastric at an acute angle in front of the arch of the aorta; it then turns round the vessel from before backward, runs up between it and the left bronchus, following the posterior surface of the aorta, which it leaves between the origin of the left common carotid artery and the subclavian to ascend to the larynx in the sulcus between the œsophagus and trachea. The right recurrent, which is shorter than the left, branches from the pneumogastric in front of the right subclavian artery. Turning under that vessel and running up behind the right common carotid, which it crosses to reach the fissure between the œsophagus and the trachea, it then proceeds to the larynx. In their course the recurrents send communicating branches to the cardiac and pulmonary nerves, and supply the œsophagus, trachea, and inferior portion of the pharynx with numerous filaments.

Rainey,* Hilton,† and Habershon,‡ from their own dissections, have described communicating filaments, running from the recurrents to the superior laryngeal nerves, while Philipeaux and Vulpian# maintain that the anastomosing fibers which are found come exclu-

* "The Recurrent Laryngeal Nerves," London "Med. Gazette," December 6, 1828.

† "Guy's Hosp. Reports," vol. ii, 1837, p. 514.

‡ "The Pathology of the Pneumogastric Nerve," "Med. Times and Gazette," vol. i, 1876.

"Sur l'anastomose qui existe entre le nerf laryngé supérieur et le nerf recurrent," "Arch. de physiol. norm. et path.," tome ii, 1869.

sively from the superior laryngeal nerves. Luschka * believed that in reality there was no communication between the superior and inferior laryngeal nerves, and that those filaments coming from the superior laryngeal which had this appearance were sometimes merely enveloped for a short distance in the same sheath with the inferior laryngeal, and at other times crossed it superficially at an acute angle to be distributed in the mucous membrane of the pharynx. Exner,† on the other hand, in one of the latest and most elaborate monographs on the innervation of the larynx, describes and pictures the ramus communicans, or "Galen's anastomosis." The relations of the inferior thyreoid artery and the recurrent laryngeal nerve are of practical importance in operations upon the œsophagus, and for the removal of the whole or a part of the thyreoid gland. Referring to Professor Dwight's ‡ paper on this subject, we find that Woelfler # stated that the nerve always passed before a branch of the artery. Kocher, ‖ on the other hand, describes the artery as passing behind the nerve, coming forward on its inner side and bending over it. Rotter △ made some fifteen examinations, and found the artery in one third as described by Kocher. Dr. Streckeisen ◊ reports the results of the examination of both sides of fifty-six bodies. He appears, however, to have had chiefly in mind the question whether or not the artery formed a loop around the nerve, and, if so, to what degree it surrounded it. It is not quite clear, therefore, in how many of his cases the artery passed before or behind the nerve. Dwight examined fifty-two bodies on both sides, and twenty-seven on one side. He found that on the right the artery was before the nerve thirty-three times, and behind it thirty-one. On the left the artery was before the nerve forty-nine times, and behind it fifteen. It appears, therefore, that the probabilities are equal that the right nerve or artery will be in front, and that on the left the chances are three to one that the nerve will be behind. Professor Dwight concludes that his series tend to support the practical deduction that, when it is necessary to tie the artery near the gland, the vessel should be carefully isolated.

* "Der Kehlkopf des Menschen," Tübingen, 1871.

† "Sitzungsber. d. Wiener Akademie d. W.," Bd. lxxxv, 1884.

‡ "Boston Med. and Surg. Journal."

"Wiener med. Wochenschr.," 1879.

‖ "Arch. für. klin. Chirurgie," Bd. xxix, 1883.

△ "Archiv für klin. Chirurg.," Bd. xxxi, 1885.

◊ Virchow's "Archiv," Bd. ciii, 1886.

It has been stated that the recurrent nerves contain sensory fibers, but we are not aware of any experimental proof to support this opinion. In our judgment, this point may be determined by the following methods:

Experiment.—Anæsthetize a dog or a cat, and fix it on its back, with its lower jaw held open, so that a perfect view of its glottis can be obtained through the mouth by throwing in light by means of an ordinary head-reflector. The animal's tongue may be held out by the fingers of the left hand, and the epiglottis raised with a long forceps by the right hand. The recurrent nerve having been exposed and *cut*, the corresponding vocal band will be seen to be completely immovable, while its fellow moves rhythmically with respiration. Place an electrode on the central cut end of the recurrent, and stimulate with currents from the feeblest to the strongest intensity; no effect whatever will be produced on the glottis. But, if we now change the electrode from the cut end of the recurrent to the trunk of the pneumogastric of the same side, and stimulate it, we do obtain a decided reflex effect upon the glottis, which was impossible when its recurrent branch was irritated. But perhaps a still more delicate proof that there are no sensitive fibers in this nerve is furnished us by the absence of a rise of blood-pressure in curarized animals on stimulation of its central end after section. The observation of Ludwig and Thiry that irritation of sensitive nerves was followed by a rise of the blood-pressure occasioned by reflex contraction of the muscular coats of the arteries led us to apply this test to the recurrent, the method employed being that described by Dittmar and Miescher in their researches * on the sensitive fibers in the spinal cord.

We have used dogs and cats for this purpose without, in a single instance, being able to observe a rise of the blood-pressure when the recurrent was stimulated with weak or strong currents, which invariably followed when the internal popliteal nerve was irritated. We subjoin the details of one of the experiments, and the curves showing the difference between the action of these two nerves on the blood-pressure.

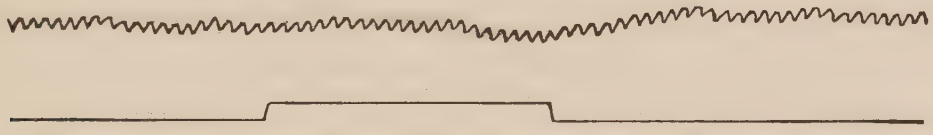
April 13, 1887.—Small black-and-tan dog. Curarized. Artificial respiration. Left recurrent and left internal popliteal nerves laid bare and cut previous to stimulation. The recording pen of a mercury manometer, which was connected with the right femoral

* "Arbeiten a. d. physiol. Anstalt," Leipsic, 1870.

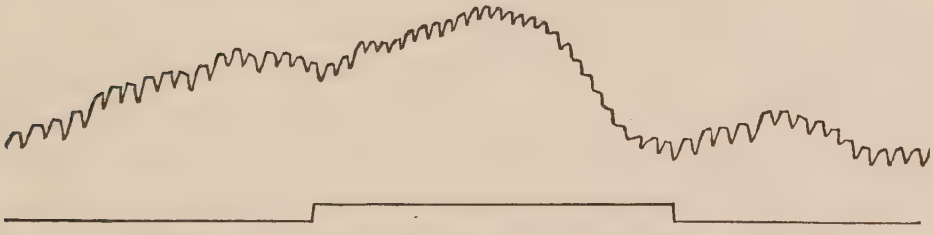
artery, recorded the blood-pressure on a long roll of paper in the usual manner.

By reading these tracings from right to left, it will be seen that irritation of the recurrent nerve with a very powerful current was not followed by any change of pressure, while, when the internal popliteal was stimulated, a marked rise was observed. We consider, therefore, that, as far as dogs and cats are concerned, the recurrents are purely motor nerves.

The recurrent nerve does not become the inferior laryngeal proper until it has reached the lower border of the cricoid cartilage. It is here about one millimetre in diameter.



Irritation of central end of left recurrent nerve. I=200.



Irritation of central end of left internal popliteal nerve. I=200.

Passing under the lower border of the inferior constrictor muscle of the pharynx, it enters the larynx behind the articulation of the inferior cornu of the thyreoid cartilage. It then divides into several branches, which innervate all the muscles of the larynx except the thyreo-cricoids. It is an occasional, but not a constant, phenomenon, that excitation of one of the recurrent nerves will cause a contraction of the vocal band of the opposite side, which tends to confirm the statement that there are connections between the terminal filaments of the recurrents in the laryngeal muscles. Such, then, being the course and distribution of the recurrent nerves, it remains now to investigate their rôle in carrying on the different functions of the larynx.

II. Physiology.

In looking through the mass of writings and experiments, from remote times to the present day, upon the physiology of the recurrent laryngeal nerves and the parts which they supply, we are struck, first of all, by the many discordant opinions that have been, and still are

being, recorded, and feel that the old aphorism, "experiment is fallacious and decision difficult," is as applicable to the nineteenth century as to the time when Hippocrates gave it utterance. Yet, when we consider the improvement in recent years in instruments of research, and reflect upon the advance and development of physical science, we may ask if it is not now, perhaps, the experimenter that is fallacious, and not the experiment.

It is, indeed, surprising that scarcely fifty years have passed since the first experimental study of questions pertaining to the larynx which approached completeness and was of real scientific value. We refer to a paper by John Reid (1838), of Scotland, the title of which, however, would not lead one to look for as much valuable work upon the nerves of the larynx as it contains. This important paper, to which we shall again refer, was entitled "An Experimental Investigation into the Functions of the Eighth Pair of Nerves, or the Glosso-pharyngeal, Pneumogastric, and Spinal Accessory,"* and was founded upon experiments which, as Romberg† justly says, may serve as models of philosophical inquiries.

We know, however, that from the earliest times the voice has enlisted the attention and speculation of physiologists, but the experiments of all, from Rufus the Ephesian down to Reid (1838), except, perhaps, Magendie's, were directed chiefly to finding out what influence the pneumo-gastrics or the recurrents had upon the function of phonation; whether the voice was lost after section of these nerves; and whether it could be regained when once taken away in this manner.

Rufus of Ephesus, Galen, Vesalius, and others found that the voice was lost after the pneumogastrics or the recurrents had been cut or included in a ligature, although less careful observers came to different conclusions; but we need not at present review in detail the investigations of these early writers, inasmuch, as we have just mentioned, as their researches had reference solely to the relation of these nerves to the voice.

To come down to more recent periods (1734), one of the earliest papers is by Martin, entitled "The Experiment of cutting the Recurrent Nerves carried on farther than has hitherto been done."‡ We find in it, however, little to justify the title, as Martin confined himself to cutting the recurrents of a sucking pig in order to observe the

* "Edin. Med. and Surg. Journal," vol. xlix, 1838, p. 109.

† "The Sydenham Soc.," vol. ii, 1853, p. 313.

‡ "Medical Essays of Edinburgh," vol. ii, 1734, p. 114.

effect on the voice, and recorded that, after the operation, "it could never give a squeak in the ordinary manner of these animals."

A more elaborate paper, by Haighton (1792), followed Martin's, on "Experiments made on the Laryngeal and Recurrent Branches of the Eighth Pair of Nerves, etc.,"* in which he calls attention to the "contrariety of opinions" that then prevailed, and adds, significantly: "This may possibly excite astonishment, when we consider that the subject is not of very difficult investigation, requiring for its accomplishment very little more than an unprejudiced mind and a hand accustomed to dissection." He determined that the recurrents were the true vocal nerves, and showed in one experiment on a dog that the voice could, "when taken away by the division of the nerves, again return." Galen, he tells us, said also that the voice "ought to return after it had been lost by experiment," owing to the communicating branches between the superior and inferior laryngeal nerves, but Haighton believed that it depended upon the reunion of the divided nerves. Magendie (1813), who, up to his day, furnished the most noteworthy experimental work on the larynx and gave a good description of the distribution of the recurrent nerves, believed, nevertheless, that all the muscles which they supplied opened the glottis, while the superior laryngeal nerves, in going to the thyreo-cricoid and transverse muscles, had exactly the opposite function, and closed the glottis. For many years a number of writers blindly followed the teachings of this great physiologist in regard to the functions of the laryngeal nerves and muscles. Hugh Ley, among others, believed with him that the recurrents opened the glottis, and that the transverse muscle was the principal closer of the organ; and in a work of much labor on "Croup" † Ley wrote considerable that is of interest to modern laryngologists, but much also that is most extravagant.

We have mentioned elsewhere ‡ that Magendie was the first who demonstrated experimentally the true action of the thyreo-cricoid muscle; and at the present day, when it is positively known that the office of this muscle in raising the cricoid cartilage on to the thyreoid is to produce longitudinal tension of the vocal bands, we can not read without a smile the reasons which Ley's fertile brain invented to account for this upward movement of the cricoid cartilage. He says

* "Mem. of the Medical Soc. of London," vol. iii, 1792, p. 422.

† "An Essay on the Laryngismus Stridulus or Croup-like Inspiration of Infants," London, 1836.

‡ "Experimental Researches on the Tension of the Vocal Bands," "Trans. of the Amer. Laryng. Assoc.," 1883.

he can trace no other purpose for it than that of throwing mucus into, and perhaps through, the chink. "This movement," he continues, "the posterior part of the ring being fixed, while the anterior is carried upward and inward, resembles that of a hoop, which will strike the shin with force when an attempt is made to raise it from its horizontal position by pressure upon its edge with the foot, or that of a basin containing fluid, treading upon the edge of which will throw its contents to a considerable height upon the limb, while the edge of the vessel itself may also strike the shin. The cricoid cartilage is thus a sort of pail, which, filled from the trachea, empties its contents into or through the glottis, and performs an office somewhat analogous to those scoops or buckets which, attached to a large wheel, help to deepen the river by drawing the earth from its bed, and, by the same revolution of the wheel, discharge their contents into an adjoining lighter for ballast or other purposes."

Here we see one of the most exquisite of the co-ordinated movements of the larynx—a movement upon which one of the fundamental laws of vocal physiology is founded—actually compared to the working of a dredging-machine and a mud-scow!

Hilton, in his "Lectures on Rest and Pain," gives a far more poetical explanation of this upward movement of the cricoid cartilage. In speaking of the motor branch of the superior laryngeal nerve, which supplies the thyreo-cricoid muscle, he says: "No matter how rapidly the nervous influence passes, it must reach the nearest point first, and that is apparently the reason why this little nerve takes so short a course to the crico-thyreoideus. It has long been my habit to regard the crico-thyreoidei as the muscles which are intended to tune the vocal instrument; and, as the instrument must be tuned before it can be played upon, so this nervous influence, first reaching the crico-thyreoidei, the vocal cords are put into a due state of tension, preparatory to the more precise and accurate influence of the other muscles acting directly and indirectly upon the vocal cords."

It was not long, however, before important light was shed upon these questions. Reid (1838), whose paper we have already mentioned, gave an accurate account of the anatomy of the recurrent nerves. He refuted Magendie's statement that these nerves moved only those muscles which opened the glottis; he confirmed experimentally Magendie's views of the action of the thyreo-cricoid muscles, and described the functions of the other intrinsic muscles, as they are now generally understood. He showed also that irritation of the recurrents of a dog closed the glottis.

Three years later (1841) Longet's* well-known paper appeared, which covered very much the same ground as Reid's, with practically the same results. Longet, moreover, agreed with Bischoff† that the internal branch of the spinal accessory was the motor nerve which presided over the tensors and the closers of the glottis, and that the dilators were innervated independently. Cl. Bernard's‡ careful experiments confirm this view. He showed that section of the spinal accessory in a kitten five weeks old was followed by aphonia, but glottic respiration remained free. Two days afterward, the kitten having remained well but voiceless, its recurrents were cut, when it instantly died asphyxiated. He concludes from this that the pneumogastric has a motor power independent of the spinal accessory which permits the animal to breathe after the latter has been cut; or, in other words, the larynx is a vocal organ when excited by the spinal accessory, and a respiratory organ when under the influence of the pneumogastric, or more probably of other motor nerves associated with it. In certain animals, as the chimpanzee, the internal branch of the spinal accessory does not blend with the pneumogastric, but goes direct as a separate nerve to the larynx.

Conclusive as were the experiments of Magendie, Reid, Longet, and others who have followed them, we find, even at the present day, differences of opinion in regard to the offices of certain of the intrinsic laryngeal muscles, about which our knowledge is apparently exact, as their function has been experimentally proved. The salient points of these questions, however, although they will always be more or less at the mercy of theorists, may be considered as tolerably well established. We know that the recurrent nerves supply those muscles which both open and shut the glottis—all of the intrinsic muscles, in fact, except the longitudinal tensors, the thyreo-cricoids; also, in part at least, those muscles which compose the ventricular bands and the ary-epiglottic folds. It is reasonably certain, too, that the internal thyreo-arytenoids, the lateral crico-arytenoids, and the transverse muscles are the closers, while the posterior crico-arytenoids are the only openers of the glottis.

In coming now to the subject-matter of this paper, we wish to express our gratification at having had the other Boston members of

* "Recherches expérimentales sur les fonctions des nerfs du larynx," etc., "Gaz. méd. de Paris," ix, 1841.

† "Nervii accessorii Willisii anat. et physiol.," Heidelberg, 1832.

‡ "Fonctions du nerf spinal," etc., "Leçons sur la physiologie et la pathologie du système nerveux," tome ii, Paris, 1858.

this association (Dr. Knight, Dr. Langmaid, and Dr. De Blois) as witnesses to some of the results herein recorded. Our thanks are especially due Dr. J. Solis-Cohen for his interest in this subject, who, with Dr. Edward Martin, of Philadelphia, was willing to submit to a long and tedious journey to pass a day of experimental investigation with us in the laboratory. It has been our object in our present series of observations, as in all our experimental work of the past five years, to multiply largely the experiments and to record only such as were typical and had been seen by other eyes besides our own. We have pursued this plan in order to eliminate, as far as possible, sources of error, and, as our work has always been undertaken without any preconceived theory to support, or care as to what might or might not happen, provided a fact could be established, we think our mistakes—if mistakes there be—will be due to some fault in our methods rather than in our observation.

In arriving at an appreciation of the physiology of the recurrent laryngeal nerves, it may be well to view them in the light of our knowledge of the functions of the organ over which they preside. We can recognize three distinct functions of the larynx controlled by three distinct groups of muscles, which are all innervated by the recurrent nerves. These wonderful nerve-trunks, therefore, which, as previously stated, are but one millimetre in diameter, contain sets of nerve-fibers as distinct as the functions of the groups of muscles which they supply. Mentioned in the order of their importance to life, these muscular groups and their functions are: (*a*) Those that carry on the respiratory function of the larynx; (*b*) the sphincter group, which serve to close the lumen of the larynx to prevent the entrance of foreign bodies, and play an important part in all expulsive acts, such as coughing, sneezing, retching, vomiting, or defecation, "or in those muscular actions where it is necessary to have the thorax fixed in order to enable the muscles attached to it to act with greater advantage or greater precision" (Lauder Brunton and Cash *); and (*c*) the phonatory muscles.†

The respiratory and the phonatory muscles which are attached to the arytenoid cartilages have diametrically opposite action. The

* "The Valvular Action of the Larynx," "Journal of Anatomy and Physiology," vol. xvii.

† It must be recognized, however, that some of the fibers belonging to the group of muscles commonly described as the thyreo-arytenoids may act as sphincters under certain conditions, while at other times they serve purposes of phonation.

respiratory muscles (the posterior crico-arytenoids) hold the glottis open for the ingress and egress of air, and, on direct stimulation, they open it still wider, while the phonatory muscles, when called into play, close the glottis by approximating the vocal bands. The vocal bands are brought into apposition in the median line of the glottis for sound production by a most delicate co-ordination of automatic muscular action, and not by forcible constriction in the sense with which we apply that word to the sphincter-like working of the muscles contained in those portions of the larynx above the vocal bands —namely, in the ary-epiglottic folds and in the ventricular bands.

These functions of the larynx can be watched in the laryngoscopic mirror. If the subject under examination takes a deep inspiration, the glottis will immediately dilate under the increased respiratory stimulus; if a probe or other instrument is passed into the interior of the organ, its walls will instantly shut tightly around it, and reflex coughing or retching will be produced; if a vocal sound is emitted, it will be accompanied by an approximation of the vocal bands. Since the recurrent furnishes these groups of muscles with nerve-force, we must speak of it, like the organ over which it presides, as having three functions, its effect upon the larynx depending upon the particular set of nerve-fibers which are called into action. If the different sets of filaments contained in the trunk of the nerve could be traced to their origin, and there differentiated and separately stimulated, we might undoubtedly produce an opening or a closing of the glottis at will, according to the function of the nerve-fibers operated upon.

But when we experiment with the nerve-trunk itself we find there all the different fibers packed together in a small compass, and on applying a current of electricity to it we can not be positive that we are stimulating all of its component filaments equally and simultaneously. We have no definite knowledge of the numerical or topographical relation between the dilating and the constricting fibers. The fact that a stimulus applied to the recurrent nerve of some animals causes a dilatation of the glottis, while in others, under the same conditions, it produces a closure, and again, in the same animal, at one moment dilatation and at another moment closure, according to circumstances, shows us what a complex nerve we are dealing with, and how great are our difficulties when we attempt to apply results obtained in the lower animals to man.

The most important function of the larynx is that of respiration. The muscles which regulate this vital act are the largest of the in-

trinsic laryngeal group. They are in ceaseless activity during life, holding the glottis open to permit the passage of air to and from the lungs. Theoretically, we should expect that the nerve-fibers which innervate these muscles would be the most numerous, the most sensitive, and of greater resisting power than all the other individual filaments of the recurrent nerve; and also that any stimulus applied to the nerve would excite a contraction of these muscles, since they are the largest of the laryngeal group, and the most bountifully supplied with nerve-force; but practically we know that this is not universally the case. If, for instance, the recurrent nerves of dogs that are unnarcotized or slightly under the influence of ether, chloroform, chloral, or morphine, are stimulated with the feeblest current of electricity, the first effect noticeable upon the glottis is a vibratory movement of the vocal bands, with a tendency toward closure, which, on gradually increasing the intensity of the stimulus, becomes a firm closure of the glottis through tetanic contraction of the adductor muscles. Now, it is very remarkable that if we irritate the recurrent nerves of a cat under the same conditions, the effect upon the glottis is exactly the opposite. The cat's glottis will dilate instead of closing. Contrary to what we observe in dogs, the earliest effect of a weak stimulus applied to the recurrent nerves of the cat is to produce a vibratory movement of the vocal bands with a tendency toward dilatation, which immediately merges into a rigid dilatation as the strength of the stimulus is increased.

Dr. Douglas Powell,* of London, has drawn our attention to the fact that the same phenomena are not observed in different animals, and he has recorded that in the cat, instead of closure, powerful abduction of the vocal bands was obtained on galvanizing the recurrent nerves.

Our own observations agree with those of Dr. Powell, and we think we may affirm that in the dog the function of the recurrent nerve, under normal conditions, is to close the glottis, while in the cat, under similar conditions, it is to open the glottis. These effects take place whether stimulation be applied to the intact nerves or to their peripheral ends after section. These opposite results in the lower animals render the practical and pertinent question, "*What is the normal function of the recurrent nerve in man?*" difficult to answer until we are able to determine which animal man most resembles, or until we subject him to direct experimentation.

The results of experiments on man have not been entirely satis-

* "Med. Times and Gazette," Dec. 19, 1874, p. 701.

factory, but they all point in one direction—viz., that stimuli applied to the nerve close the glottis. We know of but one series of experiments made on man where the recurrent nerve was dissected out and irritated. Dr. W. W. Keen,* of Philadelphia, in 1875 experimented on the recurrent nerve of a criminal recently hanged, in order to determine the question whether a chiasm of the minute fibers of the nerve existed. He says that repeated faradization, both with weak and with strong currents, of the left recurrent and left vagus produced decided movements of the left vocal cord only. The character of the movements (adduction or abduction) was not mentioned, but Dr. Keen has recently informed the writer that, although he would not like, at this distance of time, to assert positively that the left cord was adducted, he is as reasonably certain that such was the fact as one can be of a remembered fact after a long interval of time.

The attempts that have been made on the living subject to excite the recurrent nerves through the skin and soft parts of the neck, and to observe the effect upon the glottis through the laryngoscopic mirror, have been crowned with a certain degree of success, and it has always been the adductor muscles that were seen to respond to the stimulation.

Gerhardt,† the earliest investigator in this direction, reached negative or doubtful conclusions. Dr. Pauly, of Posen, and Professor Quincke‡ employed this percutaneous method to reach the recurrent nerves, and observed that stimulation was followed by complete closure of the glottis. Rossbach,# Erb,‖ and Kaplan△ have each determined that it was upon the adductor muscles of the glottis that the electrical stimulation was manifested, but, as the results were not constant, and as it was uncertain whether the effects were of a reflex nature or direct upon the nerve, or perhaps upon the laryngeal muscles themselves, we can not regard these observations as throwing any very clear light upon our present inquiry. It is noteworthy, however, that an opening of the glottis has not been recorded by those who have experimented with the recurrent nerves in this man-

* "Trans. of the College of Physicians of Philadelphia," Third Series, vol. i, 1875.

† Virchow's "Archiv," vol. xxvii, 1863.

‡ Referred to by Kaplan, "Experimenteller Beitrag zur electrischen Erregbarkeit d. Glottismusculatur," etc. Inaugural Dissertation, Berlin, 1884.

"Monatsschr. für Ohrenheilkunde," etc., October, 1881, No. 10, p. 166.

‖ "Handbuch der Elektrotherapie," Leipsic, 1882, p. 472.

△ *Loc. cit.*

ner; and, from our empirical knowledge of the action of the muscles of the glottis in health and in disease, we consider it reasonably certain that any irritation of the trunk of the recurrent nerve in man would produce a spasm of the glottis and not a dilatation.

Let us now return to the lower animals. We have already said that stimulation of the recurrent nerves of the dog, under normal conditions, *closes*, while excitation of the recurrent nerves of the cat *opens*, the glottis. Yet it is in our power, under certain circumstances, to reverse this natural order of phenomena in these two animals, and to produce in the dog a dilatation and in the cat a closure. The sole condition in the dog, so far as our present experience goes, by which a dilatation of the glottis can be obtained, is through the influence of sulphuric ether, and when the animal is in profound morphine narcosis.

From some undiscovered cause ether completely abolishes the offices of the glottis-closers. It has some unknown peripheral effect upon either the laryngeal muscles or nerves, or both, so that when a dog is in profound ether narcosis his glottis no longer closes when the recurrent nerves are stimulated, but, on the contrary, is opened widely through the action of the glottis-openers, upon which ether has, apparently, no influence.

In a paper* entitled "The Respiratory Function of the Human Larynx," published two years ago, we recorded this fact for the first time. From the date of that observation to the present time experimental researches have been prosecuted in the physiological laboratory of the Harvard Medical School, by others as well as by ourselves, with the view of determining the cause of this "ether-effect," and whether sulphuric ether would have analogous effects upon other nerves and upon other groups of antagonistic muscles.

While we were engaged in this direction, having established beyond doubt the *fact* of what for the sake of brevity we shall call the "ether-effect," a paper appeared in the "American Journal of the Medical Sciences," July, 1886, by Dr. F. Donaldson, Jr., wherein the writer stated that he had been unable to obtain the "ether-effect." He did, however, observe a dilatation of a dog's glottis when the animal was under ether, but inferred that the phenomenon was due to weak stimulation of the recurrent nerve instead of to the ether, inasmuch as he produced a closure of the glottis on increasing the intensity of the current.

Shortly after this paper was issued, Felix Semon, M. D., F. R. C. P.,

* "New York Medical Journal," July 4, 1885.

and Victor Horsley, B. S., F. R. S.,* published in a lucid report the results of their investigations on this subject, which fully verified our own observations. These authors also state that they were able to obtain dilatation with feeble stimuli, yet they call our attention to the fact, and it is here the practical point, that it was only in those cases in which the animal was not deeply etherized.

The dilatation, it seems to us, should be ascribed to the action of the narcotic.

The strength of the current, however, is a factor in determining whether dilatation or closure is produced, and it is undoubtedly correct that *in certain stages of etherization*, as we shall hereafter demonstrate, abduction may be called forth by a weak stimulus, while a stronger stimulus, with the same amount of ether, will produce the ordinary result of closure. But we think it is impracticable to use strong stimuli in these or other studies in experimental laryngology.

In our investigations on the "ether-effect," conducted two years ago, the intensity of the currents used was always of the feeblest, and it therefore struck us as somewhat extraordinary that others who followed us should lay so much stress upon the fact that weak stimuli should be the cause of the opening of the glottis, which, in reality, as we had proved, was due to the effect of sulphuric ether.

Considering, therefore, the confusion, although it appears to us to be more fancied than real, that has slightly befogged this question, we have felt it incumbent upon us to rehandle the subject.

It may be well here to explain more in detail than we have hitherto done the terms we use to describe the different shapes the glottis may assume in response to electrical stimulation of the recurrent nerves. By *complete dilatation* we mean that both vocal processes of the arytenoid cartilages are rotated forcibly outward, the vocal bands, in marked instances, appearing to lie flat against the walls of the larynx, this extreme opening being maintained as long as the stimulation is kept up. By *mixed movement* we mean an opening in the posterior portion of the glottis and a contraction of the ligamentous portion in front, giving to the glottis a rhomboidal shape. This shape appears to arise in two ways—either by an active opening behind accompanied by an active closure in front, or simply by a *want* of *closure* behind and an active closure in front. The

* "On an Apparently Peripheral and Differential Action of Ether upon the Laryngeal Muscles," "Brit. Med. Journal," August 23, 1886 p. 405.

large development of the cartilaginous portion of the glottis in the dog should be borne in mind in interpreting these appearances. The agency by which the mixed movement is brought about is not perfectly understood. It would seem, in some instances, as if all the intrinsic group of muscles were responding equally to the stimulation except the lateral crico-arytenoids and the transversus; hence the closure in front and the opening behind; but, the lateral and transverse muscles not acting, the lozenge-shaped glottis is the result. By *complete closure* we mean close apposition of the ligamentous and cartilaginous portions of the glottis.

When we consider what a complicated and delicate organ the larynx is, being made up of so many cartilages which are freely movable on each other by means of the numerous muscles attached to them, the fibers of which run in every possible direction, it need not cause surprise that there should be occasional variations in the appearances of the glottis in different dogs following stimulation of the recurrents which may be due to some peculiarity of the nerve distribution or to unusual strength of some of the laryngeal muscles.

But of the general results in dogs we think there is not much ground for doubt or for any difference of opinion of practical value. Errors of interpretation may be lessened and the most trustworthy results obtained by placing a shielded electrode on each nerve five to eight centimetres below the cricoid cartilage, the effect of stimulation on the glottis being watched through the mouth, when both nerves are irritated equally and simultaneously.

Small and young dogs, in our experience, are much more satisfactory for experimental purposes than old or very large ones. The breed seems to be of no consequence.

Our present series of observations have been conducted on forty-two dogs, and comprise three hundred and twelve recorded experiments. Of these animals, twelve have been devoted to a study of the effects on the glottis of stimulating the nerves when the dog was under chloral, chloroform, or morphia, eighteen to the "ether-effect," seven to the local effect of ether upon the nerve-trunk, and five to "exhaustion" experiments.

We have stated that the normal effect of irritating the recurrent nerves of dogs is to *close* the glottis. This statement is founded upon the investigations of others, who have experimented with and without anæsthetics, and upon our own observations with small amounts of different narcotics.

The following experiments with chloral may be taken as a type

of what occurs in the majority of cases when dogs are under small amounts of chloral, morphine, chloroform, or ether. These particular experiments are selected from a large number merely because some of our colleagues conducted the observations with us:

April 27, 1887.—Dr. Knight and Dr. Langmaid present. Medium-sized poodle bitch. Ether was first given until the femoral vein was exposed and a cannula tied into it, through which a 25-per-cent. solution of chloral was slowly injected. After this stage of the operation the animal had no more ether. The recurrent nerves were laid bare, and shielded electrodes placed upon them. The glottis was observed through the mouth. Stimulation of the nerves with the feeblest current was followed by a vibratory motion of the vocal bands, which, on increasing the intensity of the stimulus, passed into complete closure. Nothing resembling a dilatation or the mixed movement could be called forth in this dog.

Dr. Knight was present on another occasion, when a very small terrier bitch was the subject of experiment under similar conditions, and the results were identical in all respects. In no instance, in a chloralized dog, have we been able to elicit a complete dilatation, but the mixed movement has been seen in two cases. In one of these, Dr. Knight, Dr. Langmaid, and Dr. De Blois assisting, the dog was a very powerful animal, and, although the first few effects of stimulation were followed by closure, the mixed movement only was afterward called forth with all intensities. It would seem that this mixed movement was produced through a failure of the lateral crico-arytenoid muscles to respond to electrical stimulation, the other muscles acting equally. It may be that, as narcosis comes on, the lateral adductor is the first to lose its contractility, and, as the narcosis becomes more profound, the functions of the other adductors are also arrested, while the dilators hold out to the last.

Although we have not been able to produce complete dilatation with chloral or chloroform, it is not unlikely that, by pushing these drugs to extreme limits, we might, in certain cases, elicit such an effect. It is difficult to believe that sulphuric ether should be the only drug capable of calling forth abduction. We have succeeded in obtaining complete dilatation in a dog under an enormous dose (2–5 grammes) of morphine; but the condition of the dog under these circumstances can hardly be compared with that of the animal when a harmless dose of ether has been administered. It was with much surprise that, in the spring of 1885, we saw, for the first time, the glottis dilate under the effect of an irritation applied to the

recurrent nerve; but we immediately surmised the reason, which subsequent researches by ourselves and others have shown to be correct. It may render this study clearer if we briefly review the circumstances of our first observation, and how we were led to what appeared to us the only possible conclusion, that the dilatation was brought about through the influence of sulphuric ether. Our experiments, which had already been quite numerous, were generally performed on dogs chloralized by intravenous injection. This means of producing anæsthesia was almost invariably employed for prolonged experiments. Ether, however, was given in the first stage of the operation. The dog in which we first observed a dilatation was etherized for the purpose of passing a thread through the recurrent nerve, after which the incision in the neck was to be sewed up, and the animal returned to his kennel for a few days before further observation. On looking for the nerve, it was not found in its proper anatomical situation; but two small nerves were discovered near by. As it was doubtful what these two branches were, it was considered prudent to irritate them, and, if the glottis contracted, we could then be certain we had the nerve we were in search of, and proceed with the operation. But, when the nerves were stimulated, we were greatly surprised to see a forcible dilatation, instead of the customary closure. The assistant in physiology, Dr. Warren, was requested to come and witness this unusual sight. The ether sponge had been removed from the dog since the beginning of the experiment, and by the time Dr. Warren was ready to look at the glottis the animal was somewhat out of its influence. At all events, on stimulating the nerve a second time, closure was manifested, instead of the dilatation so evident a few moments before. Here we had in the same dog, at a short interval, both a dilatation and a closure of the glottis. As all the conditions were the same, with the exception of the depth of the narcosis, the natural inference was that the amount of ether in the animal must be the cause of the dilatation. A large quantity of ether was again administered, and the abduction again clearly demonstrated.

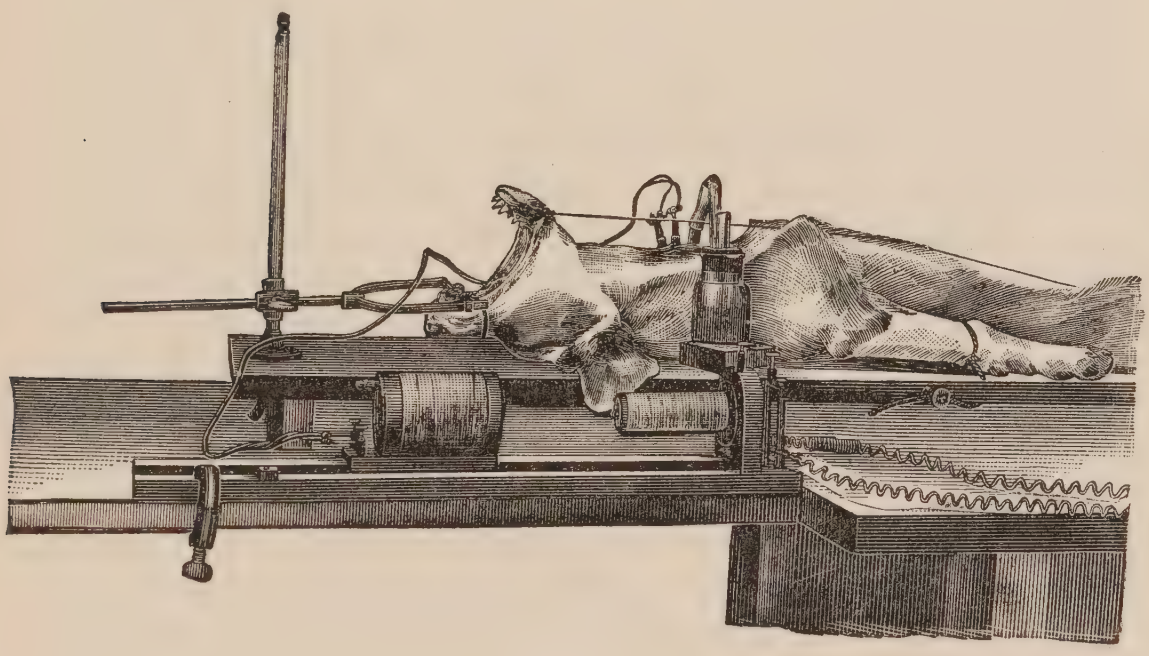
Our first idea was that the "ether-effect" was central. To test this hypothesis the recurrent was divided, and the irritation applied to its peripheral end. The glottis dilated after section of the nerves as well as before.

With regard to the fact of the "ether-effect" in dogs we have little to add. Our previous experiments were based on so many confirmatory observations on different dogs that the fact was clearly

established. The facility with which a dilatation may be elicited depends upon the susceptibility of the dog to the drug, the intensity of the irritation, and the amount given. It differs in different dogs. In the majority complete dilatation may be elicited; in other instances the mixed movement only is obtainable. In two dogs, in our experience, the effect has been negative; in these cases there was neither contraction nor dilatation on stimulation of the recurrent nerves, even in the most profound stage of etherization, but with slight anæsthesia closure took place.

"*Ether-Effect*" *Method.*—The accompanying figure will make our method clear.

The dog is here arranged in what we consider the most satisfactory manner to demonstrate the "ether-effect" in its different aspects.



It will be seen that the animal is fixed on his back, with his upper jaw tied to a dog-holder, while the lower jaw is held open by a cord fastened to the lower end of the board. A perfect view of the glottis is now made easy by holding the tongue forward in one hand and lifting the tip of the epiglottis by a long forceps with the other. Tracheotomy has been performed, and the tracheal cannula is connected with a bottle containing ether by a short piece of rubber tubing, which can readily be adjusted to or removed from the cannula. A shielded electrode is upon each recurrent nerve; the electrodes are connected with each other, and in communication with an induction apparatus, which is supplied from one Grove cell in a bat-

tery closet. The secondary coil of the apparatus is on a slide, so that it can be moved to and fro at will, according to the intensity desired. It is our custom to begin an experiment with the secondary coil so far removed from the primary that no effect is manifest on the glottis by opening the short-circuit key connected with the secondary coil, and then to slowly approach the secondary coil toward the primary until some effect on the vocal bands is produced. When a dog is very slightly etherized, the first movement noticed of the vocal bands is of a vibratory character, which changes into a closure of the glottis as the intensity of the current is increased. Now, having determined on a given dog the feeblest stimulation that will provoke a closure of the glottis with the smallest amount of ether, let us attach the ether-bottle to the tracheal cannula, and watch the evolution of the "ether-effect" through the mouth. By irritating the recurrent nerves at intervals of a few moments (the intensity of the current, be it understood, being always the same), the following changes will be noticed as the animal consumes more and more ether: The constrictors soon show signs of failure to respond to stimulation, and, instead of a closure of the glottis, the mixed movement is seen, which in time gives way to complete dilatation when the dog is sufficiently saturated with ether. By now removing the ether at this point of complete dilatation, the reverse picture of the above phenomena can be traced as the animal emerges from profound ether narcosis. The dilatation will soon give way to the mixed movement; this mixed appearance will gradually become less and less until, finally, complete constriction supervenes, when we have returned to the starting-point of the experiment. The ether may now be again administered, and the experiment repeated. Mention has already been made of individual canine peculiarities, some of the animals being very susceptible to ether, while in others the mixed movement only can be demonstrated, no matter how much of the anæsthetic is given. In presenting detailed experiments, we shall select from our note-books the most typical cases, which may be taken as representative of the average dog. The following experiment shows the time it may take to complete the circle of the "ether-effect" when the intensity of the stimulation remains the same as just described.

December 22, 1886.—Smooth-haired mongrel bitch, ten to eleven months old. Arranged for observation as previously described. Intensity of stimulation = 5, which was the feeblest current possible to produce a decided effect upon the vocal bands.

TABLE I.

	Time, A. M.	Result.
Ether applied.	10.15	Contraction.
	10.16	Mixed movement beginning.
	10.17	Mixed movement more marked.
Ether removed.	10.18½	Complete dilatation.
	10.20	Mixed movement.
	10.22	Almost complete contraction.
	10.23	Complete contraction.

It will be noticed that in this particular dog eight minutes were sufficient to exhibit the "ether-effect" in all its stages.

All our animals, we need hardly say, were etherized while being prepared for experimentation, which generally occupied from a half to three quarters of an hour. During that time the dog was given merely enough ether to keep him quiet and free from pain. Some dogs pass very quickly and quietly under the influence of ether, and come out of it with surprising rapidity, while others are very bad etherizers.

We may add another experiment here, like the one just detailed, solely for the sake of comparison, on an older dog than the last and one not quite as susceptible, but the variations are within narrow limits, as has been the case in all similar observations.

December 19, 1886.—Rough-haired terrier bitch, about two years old. Intensity of stimulation = 6, which was the weakest we were able to use, as a feebler current caused merely a vibratory movement of the vocal bands.

TABLE II.

	Time, A. M.	Result.
Ether applied.	11.13	Contraction.
	11.15	Mixed movement.
Ether removed.	11.18	Complete dilatation.
	11.19	Dilatation.
	11.20	Dilatation.
	11.21	Dilatation accompanied by slight vibration.
	11.22	Dilatation less marked; tendency toward mixed movement.
	11.23	Mixed movement.
	11.25	Almost complete contraction; on increasing the intensity of the stimulation at this point very slightly, a complete and vigorous contraction took place.

Leaving the question of the phenomena consequent upon the same stimulus in different stages of etherization, we will pass to a consideration of the effect of weak and strong stimuli rapidly applied in succession to the recurrent nerves, according to the depth of the

ether narcosis. For this purpose, let us start with a dog in the most profound state of etherization, at a point where stimulation with currents, from the weakest to the most powerful that it is reasonable to use, will fail to produce a closure of the glottis. When this condition is determined, remove the ether and proceed in the following manner until the experiment is completed: Place the secondary coil so far from the primary that there will be practically no current; then, leaving the short-circuit key open, move the secondary toward the primary coil at stated intervals, noting the different effects upon the glottis, according to the intensity of the stimulation, as the animal comes out of the ether. It will be found, under these circumstances, that dilatation or the mixed movement with *all* intensities soon gives way to dilatation or the mixed movement with weak stimuli only, and contraction with stronger; and that contraction is called forth by stimuli less and less strong as the dog becomes less and less etherized, until finally contraction only is produced by even the feeblest irritation. We submit the following table, showing in minutes the time it usually takes to demonstrate these phenomena.

November 28, 1886.—Well-bred collie bitch, six to seven months old. Arranged for observation as already described. Profoundly etherized. Ether removed at beginning of experiment, 10.39 A. M. (*a*). The intensity of the irritation necessary to produce the results indicated at the head of each column is shown by the numerals below.

TABLE III.

Time, A. M.	Vibration.	Dilatation.	Mixed.	Closure.
a. 10.39	2	5	22	....
b. 10.40	2	....	9·5	....
c. 10.41	2	....	4·8	....
d. 10.41½	1·5	....	4·8	22
e. 10.42	1·5	....	4·8	9
f. 10.42½	1·5	....	....	4·8
g. 10.43½	1·5	....	....	3

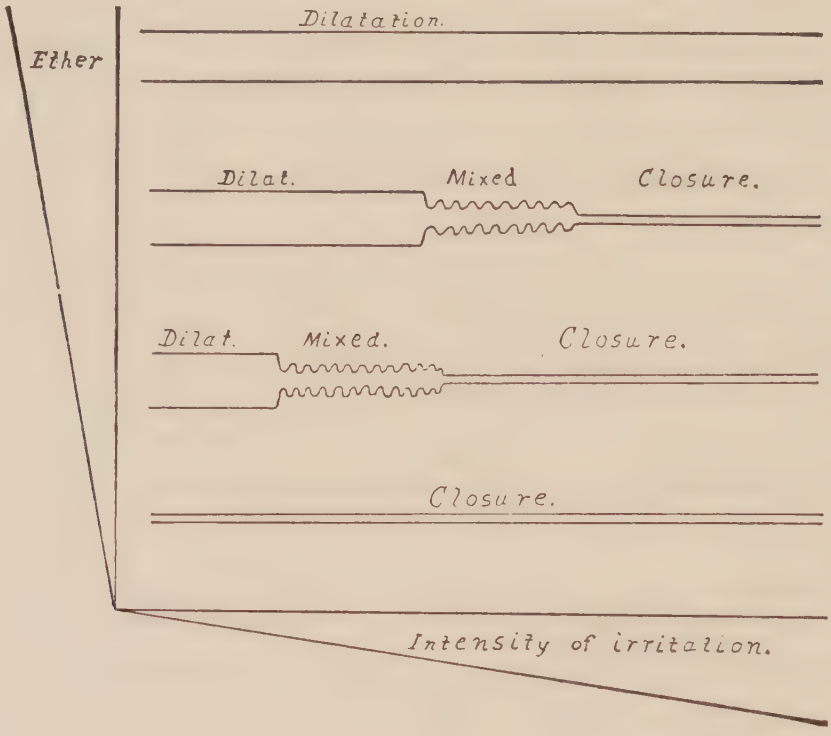
An analysis of this table shows that at the beginning of the experiment (*a*), when the dog was thoroughly saturated with ether, vibrations of the vocal bands were called forth by the excessively weak stimulus of 2; as the secondary coil was moved toward the primary, complete dilatation was manifested at 5, which persisted until the comparatively powerful intensity of 22 was reached, when the mixed movement supervened. No closure of the glottis could be

obtained in this stage of etherization, even by the strongest stimulation. One minute later (*b*), the secondary coil being again started at the farthermost point from the primary, vibrations were noticed at the same intensity as before, but dilatation was no longer obtainable, although the ether had been removed but one minute. The mixed movement, however, was seen to occur with a much feebler irritation (9·5). Still no closure. After another minute had elapsed (*c*) the mixed movement was elicited by a still feebler intensity (4·8); otherwise no change. At the next trial (*d*), half a minute later, a marked change was observed. The mixed movement remained the same, but vibration took place with a feebler stimulus (1·5), and now, for the first time, closure was effected at 22. Here it will be noticed that closure of the glottis was brought about by the same intensity (22), which two minutes and a half before (*a*) had given rise to the mixed movement, and when it was impossible to close the glottis even with the strongest stimulation. Half a minute later (*e*) closure was produced by a stimulus less than one half as powerful as in the previous trial (*d*). The other conditions remained the same. At the expiration of another thirty seconds, the dog being now pretty well out of his ether (*f*), the mixed movement was no longer manifest, and closure was easily effected by a very feeble irritation (4·8). One minute after this (*g*), the animal having so little ether left in him that he was beginning to struggle, a complete closure was called forth by an intensity of 3. At this point ether was again administered. The duration of the experiment was four minutes and a half from the time (*a*) when a closure was impossible with the strongest stimulus to (*g*) when closure took place with an excessively weak stimulus.

The foregoing method of watching the "ether-effect" is very conclusive. The experiment may be repeated on the same dog several times with almost mathematical precision, and it has been a source of surprise to us that there should be so little variation in the transitional stages of the phenomena, even in the same dog; but it is quite natural that there should be differences in different dogs. The differences, however, are only in the *length of time* necessary to bring out the phenomena with ether, not in the character of the phenomena themselves. We have devoted eighteen separate dogs to the study of this particular question of the effects of intensity of varying strengths, according to the amount of ether in the animal, and the results on a susceptible dog are identical—namely, dilatation of the glottis is obtained with *all* intensities when the dog is pro-

foundly etherized; but as there is less and less ether in the animal, dilatation is obtained *pari passu* by weaker and weaker stimuli and closure by stronger, until the normal closure is effected by *all* intensities when the dog is very slightly under the influence of the drug.

The "ether-effect" may be represented diagrammatically as follows:



The converging lines on the left represent the amount of ether, which diminishes from above downward. The diverging lines at the bottom of the diagram, as read from left to right, indicate the increase in the intensity of the irritation. Starting now at the bottom of the diagram with a very small amount of ether, the two parallel lines close together indicate a closure of the glottis with all intensities. Going a trifle higher, we find with a larger amount of ether dilatation takes place with weak stimuli, which changes into the mixed movement as the intensity of irritation is increased and finally closure supervenes. The same general effect noted directly above is observed with a larger amount of ether, only the dilatation and the mixed movement do not give way so early to complete closure as the intensity is increased. Finally, with a full amount of ether, dilatation is elicited with all intensities.

Shortly after our first observations were published (autumn of 1885 and spring of 1886) Mr. J. W. Perkins, interne at the Children's Hospital, Boston, and Dr. F. W. Ellis undertook a series of

investigations on the sciatic nerve of the frog in order to decide whether similar peripheral "ether-effects" could be elicited from this nerve, which, like the recurrent laryngeal, supplies groups of antagonistic muscles. The results of these observers have been embodied in a paper by Professor Bowditch, entitled "The Action of Sulphuric Ether on the Peripheral Nervous System," which may be found in the "American Journal of the Medical Sciences" for April, 1887. We must refer those interested in this question to the original paper for the details of the results of the experiments, for we shall quote here only such portions as are of importance in connection with our present study of the recurrent nerves, and which led to fresh experiments on our part. Perkins and Ellis found that stimulation of the sciatic nerve of the frog, treated by various methods with ether, produced opposite effects from those without ether. It was determined also that similar to the experiments on the recurrent nerve, the "ether-effect" was at the periphery, and from a study of the question, whether it was upon the nerve-trunks, the nerve terminations, or the muscular fibers, Professor Bowditch concludes that, as far as the sciatic nerve of the frog is concerned, the "ether-effect" is a phenomenon dependent upon the action of the drug upon the nerve-trunk, and that it exercises an elective and paralyzing action upon certain of the nerve-fibers. This explanation, however, of the "ether-effect" being a partial paralysis of the nerve-fibers, although justified from the studies on the sciatic nerve of the frog, is not applicable to the action of the drug on the recurrent nerves of the dog. Perkins and Ellis observed the "ether-effect" in the frog by applying the drug locally to the sciatic nerve. We determined, therefore, to pursue the same line of research in the dog. For this purpose chloralized dogs were used. Now, if the cause of the "ether-effect" resided in the nerve-fibers themselves, we might expect to obtain a dilatation of the glottis by subjecting the trunk of the recurrent to the local action of the drug. This procedure was accomplished by attaching a gutter to the shielded electrode, into which a solution of ether could be dropped directly on to the nerve-trunk, the effects on the glottis being watched through the mouth while stimulations were applied to the nerve below the point where the ether was working its local action. We have tested this local effect of ether on seven dogs without being able in a single experiment to obtain a dilatation. A six-per-cent. solution of ether was generally used. This was found to gradually paralyze the nerve in from two to six minutes, according to the dog. It was observed also

that when a nerve had become paralyzed in this manner and was afterward washed with a half-per-cent. solution of salt, the vocal band would in a few minutes regain its mobility. The shortest time that it took the vocal band to recover its motion was seven minutes and the longest twenty-two minutes. The only result of the stimulations of the recurrent nerve in chloralized dogs which had been subjected to the local action of ether was that, as the nerve became more and more paralyzed, it required a stronger and stronger stimulus to cause a contraction of the corresponding vocal band. Abduction was never obtained. We will describe one experiment in detail to show the different steps in the operations.

TABLE IV.

Time, A.M.	Intensity of irritation.	Results.	Remarks.
10.43....	2½	Contraction.	6% solution of ether applied locally to right recurrent nerve.
10.44....	2½	"	
10.45½...	2½	Contraction less vigorous; right vocal band paralyzed.	
10.46....	2½	Contraction feeble.	Necessary to increase intensity of stimulation.
10.47....	5½–7	Vibration, contraction.	
10.48....	5½–8	" "	
10.49....	7½–10	" "	
10.50....	9½–13	" "	
10.51....	10–15	" "	
10.52....	10–15	" "	More chloral given.
10.58....	12–18	" "	More ether on nerve.
11.01½...	12–20	" "	
11.03....	13–20	" "	
11.05....	15–25	" "	Contractions very feeble indeed; electrode changed to left recurrent; right recurrent washed with salt solution.
11.12....	....		Right vocal band has recovered its respiratory movements.
11.12½...	0·7–1	Vibration, contraction.	Stimulation of left recurrent; more chloral given.
11.18....	....		Ether locally on left recurrent.
11.18½...	0·7–1	Vibration, contraction.	
11.19½...	0·7–1	" "	
11.22....	7½–9	Vibration, contraction; left vocal band paralyzed.	
11.23....	7½–9	Vibration, contraction.	
11.24½...	7½–10	" "	More ether on nerve.
11.31....	9–12	" "	
11.34....	9–13	" "	More ether on nerve.
11.39....	10–15	" "	
11.40....	10–15	" "	Contractions very feeble.

January 27, 1887.—Collie dog, six months old. Ether was first administered. It was ascertained that the dog was most susceptible to the drug, and complete dilatation was obtained. The ether was now removed. The femoral vein was exposed and a cannula tied into it. Through this a twenty-five per cent. solution of chloral was slowly injected until the animal was thoroughly under its influence. The feeblest stimulus was then determined, which would produce a contraction of the glottis. This proved to be $I = 2\frac{1}{2}$ for the right side and $I = 1$ for the left; anything weaker caused merely a vibratory movement of the vocal bands. The results were as shown in Table IV.

It will be seen by this table that there was a slight difference in the behavior of the two nerves. Although the excessively weak stimulus of $2\frac{1}{2}$ (which was so feeble that it could not be perceived by placing the wires on the end of one's tongue) called forth a contraction of the right vocal band, the left responded to a still weaker current. We have not infrequently noticed a difference in this respect between the two nerves, and presume the condition of the electrodes or some slight accident in the preparation of the nerves, or the completeness with which they were freed from the connective tissue surrounding them, may account for it. But the general results were identical. Each nerve became paralyzed in from two and a half to four minutes, and, as the paralysis was more complete, it needed a more powerful stimulus to produce contraction. As before mentioned, not a trace of dilatation could be obtained. In order to show how different dogs may be affected as regards the details, though in the main the results agree perfectly, we will add here another experiment:

January 20, 1887.—Rough-haired mongrel dog, about nine months of age. Not very susceptible to ether, as the mixed movement only could be obtained. Dog chloralized.

In this dog the right vocal band became paralyzed in six minutes, and did not respond to any stimulus short of the very powerful one of 150. It regained its mobility in twenty-two minutes, and after this it responded as before (not included in the table) to weak stimuli. It will be understood that the differences are only in time and in the strength of the irritation necessary to produce the salient points in these experiments. The results were very constant and show, we think, that the cause of the "ether-effect" in dogs need not be sought in the nerve-trunks.

TABLE V.

Time, A.M.	Intensity of irritation.	Results.	Remarks.
11.07....	2–3	Vibration, contraction.	6% solution locally on right nerve.
11.09½...	2–3	" "	
11.13....	150	Right nerve paralyzed, and only responds to this very powerful irritation.	
11.16....	....		Right nerve washed with salt solution.
11.23....	....		Electrode changed to left recurrent.
11.27....	3	Contraction.	6% ether locally on left recurrent.
11.28½...	3	"	
11.29½...	3	C'ntract'ns less mark'd; vocal band becoming paralyzed.	
11.30½...	....		Left nerve paralyzed.
11.31....	80	Slight contraction.	
11.33....	80–150	Vibration, slight contraction.	
11.35....	....		Respiratory movements of right vocal band just reappeared.

We have also tested the local effect of ether on the nerve-trunk when the animal was under the constitutional influence of the drug. The only noticeable effect under these circumstances was that as the nerve became paralyzed it required a stronger and stronger stimulus to produce the dilatation.

We endeavored to determine the relative strength and endurance of the dilating and closing nerve-fibers by a series of "exhaustion" experiments conducted by applying a continuous stimulation to the recurrent trunks of dogs when the animals were under the influence of different drugs with the view of converting, if possible, the dilatation of ether into a closure by exhaustion of the abductor fibers, or changing the contraction of chloral into dilatation by exhaustion of the closing fibers. Our researches in this direction showed that it was not possible to produce a change of this sort as a result of fatigue. In no instance was dilatation changed into closure or closure into dilatation by long-continued irritation of the nerves. Continuous stimulation was kept up in different experiments for from ten to thirty minutes without altering the first effect. When the animal was kept in the same stage of etherization, dilatation lasted as long as the stimulation was maintained, while in chloralized dogs the normal closure showed no signs of weakening. In these observations

the animals were tracheotomized and the weakest intensity used that was capable of causing a well-marked effect upon the glottis.

We must here leave this subject of the action of sulphuric ether upon the laryngeal muscles of dogs with the regret that we have no explanation to offer why the glottis should dilate under its influence and contract without it. We are inclined to doubt if much light will be thrown upon the cause of the phenomenon by experimental studies on the nerve-trunks themselves such as we have instituted. The secret resides more likely in some histological or chemical difference between the opening and closing muscles of the glottis, and researches in this direction may, we trust, furnish the explanation.

In coming now to our experiments on the cat, we shall find that the normal action of its recurrent nerves is to produce an opening of the glottis instead of a closure, as in the dog. We are at a loss to account for this difference in these two animals of allied genera. Of the animals we have used for experimental purposes—namely, dogs, cats, pigs, and the horse—the cat is the only one in which dilatation of the glottis is the rule under what may be called normal conditions. We can not speak, therefore, of the "ether-effect" in the cat, since abduction takes place equally well under chloral, chloroform, morphine, or ether.

The cat may be arranged for observation in a manner similar to that we have employed for dogs. The larynx of the cat is of a much more delicate pattern than the dog's, and the vocal bands are longer and more flexible in proportion to the size of the animal.

In the living cat dilatation of the glottis is the rule, when its recurrent or pneumogastric nerves are stimulated, or their peripheral ends after section. The mixed movement, however, is sometimes observed, oftentimes at the end of an experiment, when at the beginning no contraction of either the internal or lateral muscles could be detected. But after the cat is dead the effects change. The abductions grow gradually more feeble, while the closure of the ligamentous portion of the glottis becomes more marked, until finally closure only is elicited. This difference between the action of the recurrent nerves on the glottis in the living and dead cat suggested that the condition of the blood might play a part in the phenomena, and we consequently tried the effect of asphyxiating the animal in order to see if we could bring about a closure by the deprivation of oxygen. For this purpose the cat was tracheotomized and allowed to breathe into a small rubber bag attached by a short piece of tubing to the tracheal cannula. As asphyxia was being produced the recur-

rents were stimulated from time to time, but dilatation was invariably called forth even when the cyanosis was extreme and after the respirations had ceased. In our experience with the living cat we have been able to produce a contraction of the vocal band by but one method, and that reflexly through the pneumogastric nerve. By dividing one of the pneumogastrics about the middle of the neck and stimulating the cut end nearest the brain (care being taken not to include the sympathetic), stoppage of the respiration will occur and the vocal band of the opposite side will come to a standstill in the position of expiration; on increasing the intensity of the irritation a decided contraction of the vocal band will take place, it coming in contact sometimes with its fellow. The results of our observations, which have been made on twelve cats, were quite constant, the differences being not in the character of the phenomena, but, as in all experiments on different larynges, in the extent or degree to which the effects are manifested. We subjoin the details of specimen experiments:

February 17, 1887.—Male cat. Etherized; secured on its back with mouth held open; glottis observed through the mouth; pneumogastric and recurrent nerves laid bare; irritation of the trunks of both nerves with stimuli of varying strengths produced dilatation of the glottis; same effect followed stimulation of the peripheral ends of pneumogastrics and recurrents after section.

Solution of chloral injected into external jugular vein; stimulation of peripheral cut ends of both recurrents with all intensities produced dilatation.

February 20, 1887.—Male cat. Chloroform first given, afterward chloral. Experiments of February 17th repeated with like results. Left vocal band became paralyzed from some unknown cause. In twenty minutes it began to regain its mobility, the left recurrent meanwhile having been washed with a half-per-cent. salt solution; medulla destroyed; stimulation of cut recurrents immediately afterward gave dilatation.

February 22, 1887.—Male cat. Tracheotomized; ether given through tracheal cannula; stimulation of uncut pneumogastrics and recurrents followed by dilatation; left pneumogastric cut; stimulation of its central end causes a contraction of the right vocal band. Toward the end of the experiments on this animal it was noticed that complete dilatation had given place to the "mixed" movement, there being well-marked contraction of the anterior portions of the vocal bands.

February 24, 1887.—Female cat. Chloroform, later chloral; stimulation of uncut pneumogastrics and recurrents as in previous experiments; mixed movement was observed to follow dilatation after the nerves had been irritated many times; left pneumogastric cut; irritation of its central end produced stoppage of the respiration, the vocal band on the opposite side (the right) coming to a standstill in the median line.

This cat died suddenly without apparent cause; immediately after death, stimulation of the recurrents produced the mixed movement as before; the abductions, however, soon grew very feeble, and in twenty minutes were no longer elicited by stimulating the nerves, while the contractions were still noticed, though less vigorous.

February 27, 1887.—Male cat. Killed at 10.45 A. M.; a few moments later dilatation was called forth by feeble stimuli applied to the pneumogastric and recurrent nerves; the recurrent nerves were now stimulated alternately. At 10.55 irritation of the left recurrent with weak currents produced the mixed movement, which was replaced by complete contraction on increasing the intensity of the stimulus. After this no sign of abduction could be detected, and by 11.15 the contractions had also ceased to be manifested even by the strongest irritations. Stimulation of the right recurrent at 11 produced abduction, which changed into the mixed movement on increasing the intensity of the stimulus. At 11.10 abduction had disappeared; feeble irritation caused slight contraction, which became very marked as the intensity was increased. At 11.20 only very powerful stimuli (100 to 150) produced feeble contraction as the nerve was practically exhausted. The closure after death is more marked in some cats than in others, but the difference from the dilatation during life, with one exception, has been very striking.

This one exception was observed in a cat that had been the subject of one of our asphyxia experiments, and, as the failure to contract occurred on one side only, it may be put down to some accidental cause.

April 15, 1887.—Asphyxia experiment. Female cat. Ether, afterward chloral; tracheotomized; small rubber bag attached by a piece of tubing to tracheal cannula; asphyxia begun at

10.40 A. M. Stimulation of left recurrent produced abduction of left vocal band.

10.42. Respirations fast and violent; tongue and mucous membrane of mouth becoming blue; abduction.

10.45. Respirations much slower; cyanosis extreme; abduction.

10.46. Respirations have almost ceased; abduction.

10.46½. Respirations have ceased; bag removed; glottis is tightly closed, which, when asphyxia was coming on, was widely open; stimulation of left recurrent, as before, caused dilatation of the left vocal band; in a moment or two the cat's normal respirations recommenced; a few minutes later this experiment was repeated.

10.52. Asphyxia begun; stimulation of left recurrent; abduction.

10.53. Respirations 84 to the minute; abduction.

10.54. Respirations much slower; cyanosis extreme; abduction.

10.55. Respirations have ceased; glottis closed; feeble irritation failed to produce abduction as in the previous experiment; on increasing the intensity a slight opening was observed; artificial respiration was used to resuscitate the animal without success, and the cat may be said to have died at 10.55. We then proceeded to obtain the closure after death, as in previous cats.

10.59. Glottis no longer shut; the vocal bands appear to be a little nearer the median line than the cadaveric position; stimulation of left recurrent gave contraction in anterior portion of vocal band and dilatation of the posterior part (mixed movement).

11.06. The same.

11.09. The same.

11.11. The contraction not so marked; dilatation as before.

11.13. Dilatation; no contraction noticed even on increasing the intensity of the stimulation.

11.16. Feeble dilatation.

11.18. Very feeble dilatation, practically gone.

11.20. Electrode changed to right recurrent; stimulation with feeble and strong stimuli produced contraction; no sign of abduction; the contractions grew gradually feebler, but were elicited for twenty minutes, when the nerve became completely exhausted.

April 17th.—Large male cat; chloralized; irritation of recurrents produced complete dilatation; tracheotomized and arranged for asphyxiation as in previous experiments.

11.05 A. M. Asphyxia begun; abduction.

11.07. Respirations rapid; cyanosis beginning; abduction.

11.09. Respirations about the same; cyanosis marked; abduction.

11.11. Respirations slow; glottis open; extreme cyanosis; abduction.

11.11½. Respiration has ceased; bag removed; glottis closed; abduction; artificial respiration restored the normal respiratory

movements in a few moments; this experiment was repeated three times on this animal with like results.

Experiment IV.—Cat breathing naturally; stimulation of recurrents and vagi produce abduction; left vagus and left sympathetic cut; stimulation of central end of vagus stops the respiratory movements, the vocal band of the opposite side (the right) stopping in the expiratory position. On increasing the intensity of the irritation, the contraction of the right vocal band was very marked, the right arytenoid cartilage coming sharply against the left.*

Experiment XXI.—12.08. Medulla destroyed; stimulation applied alternately to recurrents gave abduction.

12.14. Mixed movement; decided contraction of the anterior portions of the vocal bands, which was not noticeable before death.

12.20. The same.

12.25. Contraction in front; abduction behind very feeble.

12.30. Contraction both sides marked; abduction of right practically gone; of left very feeble.

12.35. Abduction entirely gone; feeble contraction still called forth with strong stimuli.

We may summarize the experimental results of this paper as follows: 1. The recurrent laryngeal nerves are purely motor in their function. 2. Their action upon the glottis differs in different animals. 3. In the dog their normal action is to close, while in the cat they open, the glottis. 4. In the dog, sulphuric ether and huge doses of morphine reverse the normal action of the recurrent nerves; the glottis, under the influence of these drugs, dilates instead of closing on stimulation of the recurrents. 5. The "ether-effect" varies according to the susceptibility of the dog, to the amount of the drug consumed, and to the intensity of the irritation. 6 Under small doses of ether, stimulation produces in dogs two effects upon the glottis, (1) vibration; (2) closure. Under larger doses, according to the intensity of the irritation, four effects may be observed: (1) vibration; (2) complete dilatation; (3) mixed movement; (4) closure. 7. Under very large doses of ether, closure, in certain dogs, can not be obtained with any intensity. 8. Under small doses of chloral, chloroform, and morphine, stimulation of

* Stimulation of the central end of the sympathetic increased the respiratory and heart-movements, and produced a great fluttering of the right vocal band, which was apparently due to the respiratory efforts. The vocal band was forcibly abducted, but did not remain fixed; it kept "fluttering" in and out, but the outward movements were the more pronounced.

the recurrent nerves of dogs is followed by the same results as under small doses of ether, namely: (1) vibration; (2) closure. 9. The mixed movement is occasionally seen in dogs when narcotics other than ether are given in large doses. 10. In the cat death reverses the normal action of the recurrent nerves. In from five to twenty minutes after death, stimulation causes a contraction of the glottis, while in the living cat dilatation is produced.

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THE EFFECT OF PURE ALCOHOL ON THE REACTION TIME, WITH A DESCRIPTION OF A NEW CHRONOSCOPE.

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The experiments which I shall report in this paper were undertaken with a view of determining the part played by pure alcohol in the changes of the reaction time under the influence of wine which the researches of Exner[1] and those of Von Vintschgau and Dietl[2] had already made known to us. A similar investigation has been carried on by Kraepelin[3] but it was recorded in Wundt's "Philosophische Studien," which I am not in the habit of seeing, so that I unfortunately did not know of his work until my own observations had been completed. As the method I have employed differs somewhat from those of my predecessors I venture to hope that my results may prove to be not altogether uninteresting.

The immediate occasion of this research was the invention by Prof. Henry P. Bowditch of a new and very convenient apparatus for measuring short intervals of time—and its construction in the workshop of the Physiological Laboratory of the Harvard Medical School. I happened just then to be particularly interested in the physiological action of alcohol and thought it an excellent opportunity to use the new appliance for recording reaction times. This apparatus has not yet been described in print and I am permitted to explain its construction and working here. It grew out of an effort to adapt the record of a vibrating tuning fork to the registration of very brief periods of time. As the earlier form of the apparatus had some resemblance (although remote and independent) to one proposed several years ago by Exner[4]

[1] S. Exner: "Experimentelle Untersuchung der einfachsten psychischen Processe." *Pflüger's Archiv f. d. ges. Physiol.* VII. 601.

[2] M. J. Dietl u. M. von Vintschgau: "Das Verhalten der physiologischen Reactionszeit unter dem Einflus von Morphium, Caffée und Wein." *Pflüger's Arch.* XVI. 316.

[3] E. Kraepelin: "Ueber die Einwirkung einiger medicamentöser Stoffe auf die Dauer einfacher psychischer Vorgänge—Zweite Abtheilung: Ueber die Einwirkung von Aethylalkohol." Wundt's *Philos. Studien* Bd. I. 573.

[4] Exner: *op. cit.* p. 643, 659.

I have adopted his name for this instrument and propose to call it the Bowditch Neuramoebimeter[5] (or nerve-reply-measurer) although its application will be seen to be much more general than the name would imply.

The details of structure are easily made out by an examination of the photographic reduction in Figure 1. It will at once be seen that

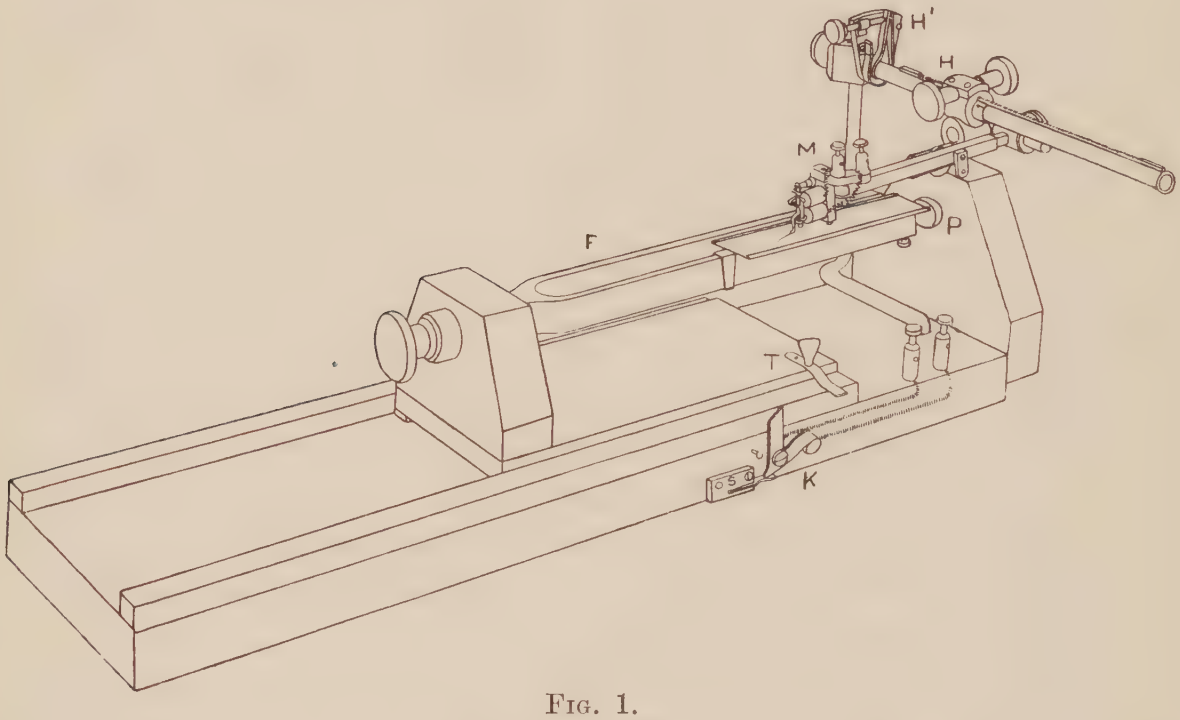


Fig. 1.

the apparatus not only has the advantage of convenience and great simplicity, as compared with most chronoscopic appliances, but also has the special merit that its expensive portions are three independent instruments which every well-equipped laboratory should possess for ordinary work and which can at any time be detached for use in other ways.

These three instruments are: 1. The standard tuning fork (F); 2. The delicate recording magnet of Deprèz (M); 3. The adjustable holder (H,H′) which in some form is almost indispensable for work with Marey's drums or similar recorders. The tuning fork carries on one arm a little brass plate whose edges are turned up to hold a strip of

[5] Νεῦρον—ἀμοιβή (a return, answer)—μέτρον—Exner had made this instrument to meet the special requirements of an investigation which Obersteiner wished to undertake with insane persons. In the report of this work[a] an instrument is pictured which is practically the same as the one described by Exner. Obersteiner prefers to call it a Psychodometer. This seems an unprofitable reduplication of names.

[a] H. Obersteiner, jr.—"Ueber eine neue einfache Methode zur Bestimmung der psychischen Leistungsfähigkeit des Gehirnes Geisteskranker. *Archiv für pathol. Anat.* 1874, lix. 427.

smoked cardboard (115 × 28 mm.) the other arm being balanced by another brass plate which is held in place by a screw clamp[6]. The fork is attached in the usual manner to a wooden carriage which slides in grooves on the larger base board. This board has an upright block at the end, held in place by a large screw which permits some movement for adjustment. In the centre of the block is an elliptical plug (P) or spreader which can be set by a rod at the back. This plug is so placed as to allow the fork to be pushed up to the head board when the long axis is perpendicular. If the fork be pulled with the spreader in this position the record is a straight line. Turning the spreader through an angle of 90° forces the prongs apart and the fork begins to vibrate when the pull removes it from the plug, the record changing from a straight line to an undulating one as seen in the sample card (Fig. 3, p. 316). At the left a brass rod runs up to carry the adjustable holder which in turn carries the writing magnet. The ease with which the magnet can be adjusted to write on the card or be lifted from it is so obvious from the plate as to call for no further description.

On the base board is seen a key (K) to which wires go from the binding posts and which may be opened by the brass strip or tongue (T), whose position on the slide can be varied by the set screw. To ensure a good electrical contact the key is faced with platinum and has a small spring (S) to keep it open or shut as the case may be. Evidently the entire arrangement for mounting and using the fork and magnet is so simple that a very moderate ability to use tools will suffice for its construction.

The working of the instrument is equally simple. A card suitably smoked is placed upon the fork as it stands drawn away from the magnet. The key (K) is opened and the plug (P) turned so as to have its long axis perpendicular. Then the magnet is lifted by pressing on the spring (H′) and held while the fork is pushed home. The magnet

[6] Such an adaptation of the tuning fork is not new. Its earliest application to physiological work appears to have been made by Klünder[a] at the suggestion of Hensen. As Klünder himself points out, the physicists had recommended its use at a still earlier date. The reader is probably most familiar with the method from the investigations of Landois[b] who used it for chronographic measurements of the pulse and heart beat.

[a] Klünder, "Voruntersuchungen über den zeitlichen Verlauf der Muskelzuckung." *Arbeiten aus d. Kieler physiol. Inst.* (1868). Kiel, 1869, p. 107. An abstract is given in the *Centralbl. f. d. med. Wiss.* 1870, p. 409. See also Gscheidlen's *Physiologische Methodik*, p. 609.

[b] Incidentally mentioned in the *Deutsche med. Wochenschrift*, 1878, IV. 348 (Landois, Ueber die Bestimmung der Fortpflanzungsgeschwindigkeit der Pulswellen und der zeitlichen Entwickelung der Rückstosselevation der Pulscurven). Details and curves may be found in several articles by Landois in Eulenburg's "*Realencyclopädie der gesammten Heilkunde*" [Graphische Untersuchungsmethoden, Herzstosscurve, &c.] and in the various editions of his *Textbook of Human Physiology*.

now drops on to the card and is adjusted, the plug is turned to spread the tuning fork, and the key is closed. While the left hand holds the head board the right pulls the fork which records its vibrations by the scratcher of the writing magnet, and also in passing opens the key (K) when the tongue (T) reaches it. We shall have then a record of the vibrations of the tuning fork whose legibility will depend on the speed with which the fork is pulled. If we connect the wires from a battery with the writing magnet and the binding posts in such a way that the key will break the circuit, we shall be able to indicate the instant of opening the key in the record; for the magnet will lose its magnetism and the pen will change its position, and this will cause a change of level in the vibrations recorded by the tuning fork. If after a brief interval a current of electricity should pass again through the magnet the pen would return to its former position, and another change of level in the record of vibrations would occur; the number of vibrations from the beginning of the first change of level to the beginning of the return gives us the measure of the time which elapsed from breaking the circuit until it was closed again. In the apparatus described a standard tuning fork (100 vibrations to the second) is used. The load changes the rate of vibration somewhat and for exact time measurements a comparison must be made with some other standard (pendulum). For the work to be reported in this paper the value of one vibration was changed from 0·0100″ to 0·01115″.

Obviously the opening of the key (K) may be adapted to giving a variety of signals dependent upon breaking an electric current (or the key may be used as a shunt for short circuiting) and we may thus signal to any of the senses of the percipient, or stimulate a nerve or muscle directly, and a reply may be given by any object which undergoes such changes on account of the stimulation as to cause an electric current to pass anew through the writing magnet. The application of the Bowditch Neuramoebimeter is thus seen to be very extended.

Although this instrument is not quite so simple as that of Exner and Obersteiner, it has certain very important advantages besides a greater variety in its applications. Exner's instrument records the movements of a vibrating rod on a plate drawn past it below, and the reply is given by pressing a knob on the projecting end of the rod and thus lifting the recording end off the plate. The percipient must then be always in the immediate neighbourhood of the instrument. He will accordingly inevitably be forced to hear a great variety of sounds incident to working the apparatus so that any one of these may serve

to complicate the signal. A well-trained and very circumspect experimenter can probably learn to neglect all sounds and attend only to the proper signal, but my own experience convinces me that the difficulty is really a very serious one. As we shall see presently the Bowditch arrangement is not absolutely free from this objection but we shall also learn how it may be reduced to a minimum.

The experiments for studying the influence of alcohol recorded the time which elapsed between the stimulation of one or two fingers of the left hand by an induction shock of moderate, but never painful, intensity and the closing of a simple key, like the ordinary telegraph key, by the right hand which rested upon it. The arrangement of the apparatus for this purpose is seen in the accompanying diagram, Fig. 2. The galvanic current from the battery (B) runs through the

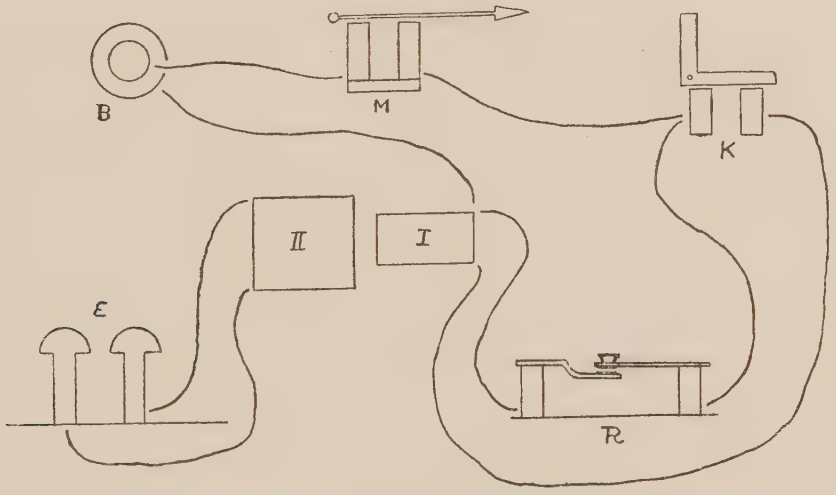


Fig. 2.

magnet (M), the key (K), and the primary coil (I). When the key is opened the induction shock goes from the secondary coil (II) to the electrodes (E) and the closure of the reply key (R) reanimates the magnet. The electrodes and reply key may be at any distance from the recording apparatus (always in a closet or in another room in these experiments) and the induction apparatus may be near either of the persons engaged in the experiment. One good (Grove) cell is sufficient. For different positions of the induction apparatus other and more economical arrangements of the wires will readily suggest themselves. It is however particularly desirable not to send the reply current through the primary coil as it produces an unpleasant and confusing shock.

The record obtained in such work is shown by the sample card reproduced in Fig. 3. These replies were given by an inexperienced

person and are printed merely to illustrate the form of legible record for the various pulls which the fork may receive. The last reply is probably in answer to something other than the induction shock—it is evidently rather too quick. For the sake of clearness much space is wasted. After a little practice it is easy to record legibly from seventeen to twenty reaction times on one card and with such speed as to fill four

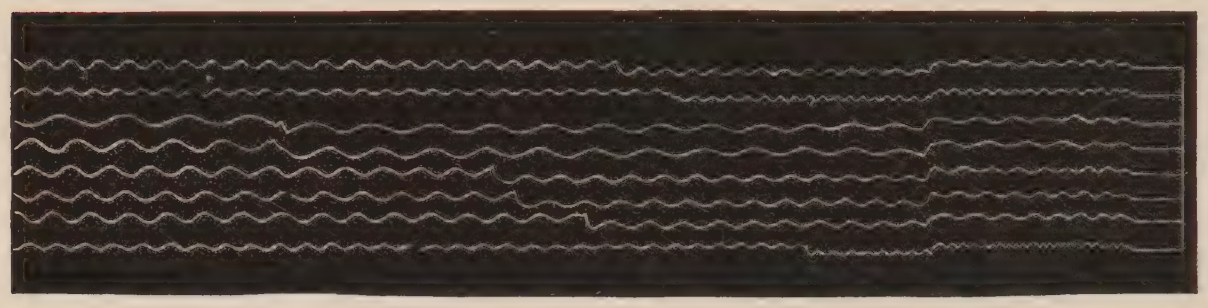


Fig. 3. Sample of Record. Full size.

cards in about ten minutes. This rate of working also allows time for the percipient to make a memorandum of the goodness of each reply. The cards are numbered and dated and then varnished. The counting may be done at any time for the record is permanent. The size of the cards is such that a large number fill but a small space; indeed it seems to me no small recommendation of the Bowditch Neuramoebimeter that the cards belonging to this paper (recording 8630 reaction times) occupy a box somewhat smaller than this printed page and only 3 cm. deep. When the vibrations are counted they can be noted on the back of the card together with the memoranda made at the time of the experiment. It is better to keep all records in vibrations, and to translate these into seconds when the necessary averaging has been done. In counting it is easy to estimate tenths of a vibration unless the pull has been a very slow one.

There is one source of danger in using the instrument before us which ought to be mentioned, that is the twang of the fork when it leaves the plug and begins to swing. This is sometimes heard in the adjoining room with great distinctness and, if the pulls be made with much regularity as to speed, may act as a confusing signal. This danger is lessened by placing the Neuramoebimeter on a large folded towel, but it is still better to pause for a varying length of time after the fork has left the plug, and then continue the pull before the vibrations are too small to give a good curve. In this way the twang becomes a part of the signal to be ready for the shock and is not confused with it.

So important is this influence that I found myself obliged to remove a large number of the earlier records (as "quick pulls") where I feared that this complication of signals might have arisen. Merely deadening this sound will not suffice, for the influence of any drug we wish to study may be to quicken the perception of the twang and thus unwittingly introduce an error. It would therefore be better to meet the difficulty in the way just suggested. Here, as in all similar work, it is important to avoid any very great regularity in taking the records. Such a habit is but too easily formed under the influence of the natural desire to work rapidly. Other observers have also called attention to this difficulty.

The general course of an experiment[7] was as follows: At some convenient hour four or five cards were filled with reaction time records; after a suitable or convenient interval another "series" of such records was taken and still later a third or fourth. On the days when alcohol was used the desired dose was taken at a varying interval before the second series of reaction times was recorded. In a few experiments a second and once a third dose of alcohol was taken. I shall accordingly speak of "normal" and of "alcohol" series but it will be observed that each day's record begins with a normal series which we may call the "normal of the day." Doubtless greater regularity in the intervals and in the doses of alcohol would have been more desirable and I should strongly urge the adoption of such a course. For these experiments I can only say that we had to work when we could find the time. The intervals between the different series were filled up with as little work as possible where they were too long to be passed in complete idleness, and so far as I know nothing was undertaken which could be reasonably supposed to influence the reaction time in the series which I shall consider.

As we wished to know the action of pure alcohol we made use of Squibb's "absolute alcohol," the purest and best which the pharmacists could furnish. As it would be practically impossible to keep this "absolute" alcohol unchanged, a certain amount of it was mixed with distilled water to make a liquid containing 25—40% (by volume) of alcohol as determined by an araeometer. This supply served for a few experiments and was then replaced by a fresh mixture. At the time of

[7] I am under great obligation to Dr H. S. Durand, then a student of the Medical School, and to another student, C. H. Abbot, D.M.D., of Berlin, Germany, for their generous and careful cooperation in these tiresome experiments. I am also indebted to Dr. F. W. Ellis who kindly took many of my own records for me.

the experiment more distilled water was usually added, together with some cane sugar to render the drink less disagreeable; at the best it is an exceedingly unpalatable combination whose consumption does not heighten the pleasure of such investigations. That the mixture is effective I can testify from personal experience. The tables which will be given later will show that the alcohol was taken at a time of day when the condition of the stomach might be expected to be such as to favour fairly rapid absorption. I may add that we were all accustomed to the moderate use of alcoholic beverages but not in the habit of drinking anything of the kind at the time of day when these experiments were made. Unfortunately no teetotalers were available for the research. The amounts of alcohol employed were such as would probably produce a distinct effect, which must necessarily vary with the individual, but we made no effort to examine the problems associated with drunkenness.

Although we used alcohol of 26—41% (again diluted somewhat as already mentioned) I shall state the amounts in equivalents of 10% alcohol. It will thus be easy to know the quantity of absolute alcohol employed or to judge of the amount as about equivalent to a moderate claret or to champagne.

Remembering that the ordinary champagne bottle of commerce holds some 800 c.cm., it will be seen from the tabulation that we have three experiments with the equivalent of $\frac{1}{7}$—$\frac{1}{5}$ of a bottle of champagne (with regard to alcohol); eleven with $\frac{1}{4}$—$\frac{1}{3}$, and seven where about $\frac{1}{2}$ a bottle or rather more was taken in one dose; there are also three cases where somewhat more than half a bottle was taken in two doses, and one in which the equivalent of nearly a whole bottle of champagne was consumed in three doses. With the exception of one experiment (where the alcohol was sipped in portions) the entire amount was drunk almost or quite immediately at the time noted. I shall give such memoranda from the note-book as will serve to indicate the alcohol effect as it manifested itself to us, and I may say in addition that I think that some effect was evident, at least to the taker, in every instance. Besides the ordinary and familiar phenomena of alcoholic stimulation we met frequently with that condition of restlessness which other observers have noted. The reply key was often closed with audibly increased vigor and the person at work thought his replies not only quicker but better than usual, although as we shall see he was not infrequently mistaken. Yet on the whole I do not think that any very marked alteration of the judgment concerning single replies was observ-

able. Personally I found that the time seemed interminably long, and had the feeling that I was hampered in some way and that the replies must be slow or at least ought to be quicker. Still as I knew from the work of others that such a feeling would probably manifest itself and that such errors were commonly made, I was doubtless rather too introspective and alcohol probably aggravated this condition.

Records of this nature may be tabulated in several ways. We may simply add them up and dividing by the number of observations get the average. For some purposes it is better to group them. If in counting we avoid noting any record as containing exactly five-tenths of a vibration (for example, reading 13·5 as 13·4 or 13·6 as the case may be) we may group all observations between 13·6 and 14·4, for example, as 14, all from 14·6 to 15·4 as 15 vibrations, and so on. We shall thus get a tabulation which gives a very good and simple view of the run of the records, and we can judge of their character better and much more easily. This form of tabulation has a still further advantage in enabling us to examine the probable error of the series with greater convenience than we could otherwise do, and I shall speak of records treated in this way as "grouped."

Before the mean of a series could be obtained by either method a certain amount of correction had to be made. Out of the 8630 records (exclusive of about 600 taken for practice) 147 proved to be mechanically defective and 15 more were so ridiculously small as to be obviously incorrect. I then also removed all the records where I myself had given a "quick pull." I felt sure that in some cases the hum of the instrument had confused the signal, and therefore rejected 231 such uncertain records regardless of the quality of the replies, and afterwards used a slow pull in all cases. Unfortunately no such precaution could be employed in my own reaction times which were taken by several observers, and I was unable to do anything more than use the quick pulls as in some measure furnishing an occasional corroboration of other evidence against the record. Besides these I also rejected from the beginning all records (191 in number) which are called "bad" or "slow" in the note book, regardless of their apparent excellence. It seems to me fair and proper to give the percipient the benefit of such doubt as he has deliberately expressed, and it is also altogether impracticable to lay down any rule as to what should be retained. The effect of alcohol on such judgments we may consider later. On grouping the 8046 records which then remained in the various series it was found that 122 "good" observations were obviously so far removed from

the rest of their own series, when grouped, as to appear valueless. This seems much better than the method of rejecting any record which is somewhat removed from those just about it in the order of taking, as has usually been done, especially in experiments involving a rather small number of reaction times. In any case there is an opportunity for the exercise of great reserve in making the removals, but as I look over the figures published by other observers I can but feel that the plan I have followed of treating each series as a whole and grouped gives more trustworthy results, since replies which seem rather far off from their immediate neighbours as taken are found to have many companions in a large series of sixty or seventy observations.

We have then finally 7924 records which are available for comparison with reference to the influence of alcohol upon the reaction time. These will be tabulated in two ways. The average obtained by dividing the sum of all the observations of a series (without any grouping) by the number of such observations I shall call the "absolute" average, and speak of that which results from the grouping as the "grouped average," the latter being obtained by multiplying the number of observations in each group by the value of the group and dividing the sum of these products by the total number of observations. This treatment not only furnishes a good method of controlling the blunders incident to the comparison of so many numbers, but also makes it easier to work out the probable error[8]. As would be expected the "grouped average" is not exactly the same as the "absolute average" and the probable

[8] The Probable Error is obtained by the formula of Dienger:

$$r_0 = 0{\cdot}6745 \sqrt{\frac{\Sigma x^2}{n(n-1)}}$$

i.e.

$$= 0{\cdot}6745 \sqrt{\left[\frac{(m-a)^2+(m-b)^2+\ldots\ldots}{n(n-1)}\right]}$$

where m is the average of n observations whose individual values are a, b, c, d.....

Knowing the value of the probable error of two sets of observations we can judge of their comparability. Thus if we wish to compare two series whose averages are M and N with the probable errors p and q respectively we may not infer that the result N really differs from M (has been brought about by the influence of alcohol for example) unless we can show that $M-N$ is greater than $p+q$, for otherwise the probable values of the two results (or their range) will overlap each other to a greater or less extent.

Again, if we compare two results and wish to know the amount of change which a certain influence has produced it is not enough to take the difference $M-N$ as expressive of this change, for M may have any value between $M+p$ and $M-p$, and similarly N is to be taken as $N \pm q$. The change then taken absolutely is $M-N$, but it has a "Range" (as I shall call it) depending on the probable error and obtained by the formula $M-N \pm (p+q)$, and I shall use this "Range" in indicating the alcohol effects by means of diagrams.

error of the one is not exactly the probable error of the other, but there is a sufficient correspondence to enable us to draw some conclusions as to the justness of such comparisons as we shall wish to make. In such curves as I shall use to express the results of these experiments dots will indicate the position of the absolute averages (or of their differences) while the probable errors (or the "ranges" due to their influence) will be shown as lines, and the position of the "grouped averages" (or of their differences) is of course at the middle of such lines.

The following tables will give the reader the necessary details of each experiment. It should be remembered that we all had some practice with the instrument before these series were begun. It will be convenient to indicate the work of each day by a Roman numeral and the series of the day by letters of the alphabet in regular order, and this designation will be used in all later tables or diagrams so that a reference to these three tables for particulars will be easy. The first column, then, gives the running number of the series—such a series consisting commonly of 4 cards or "sets" which are not noted in detail. In the second column is noted the time at which the record was made, each series requiring about 10 minutes. In the next space will be found the number of such "good" observations as were finally retained. The fourth column contains the "absolute" average[9], while the "grouped" average and its probable error form the fifth and sixth columns. In the last space finally we have the date and the nature of the day's work, the amounts of alcohol (expressed in equivalents of 10% alcohol) and the time at which it was taken, together with such other memoranda as seem necessary to an explanation of the experiment—or to give the general effect of the alcohol so far as noted at the time of the experiment.

[9] As I cared little for the absolute values of the reaction time I have not thought it worth while to correct these averages in accordance with the opening and closing error of the magnet used. The correction is about 0·0010″ and obviously does not affect the differences with which we shall have principally to do below.

TABLE I.

Experiments with A.

Number of series	Time	No. of observations (corrected)	Absolute average	Grouped average	Probable error	Remarks
I *a*	11.0	32	0·1456″	0·1450″	± 0·0025″	**15 Nov. 1885. Normal.** The time given is in all cases the minute when the series begins. The number of observations is small (as in several later days) on account of the removal of 44 "quick pulls."
I *b*	11.30	36	0·1414″	0·1415″	± 0·0016″	
I *c*	12.0	39	0·1486″	0·1478″	± 0·0017″	
II *a*	3.30	45	0·1483″	0·1484″	± 0·0014″	**17 Nov. 1885. Alcohol. At 4.0. 224 c.cm.** During the *b*-series there was a feeling of prompt reaction and one set of it was correctly recognised as especially quick.
II *b*	4.20	40	0·1430″	0·1433″	± 0·0013″	
II *c*	5.0	47	0·1567″	0·1561″	± 0·0016″	
II *d*	5.41	36	0·1541″	0·1545″	± 0·0015″	
III *a*	4.10	43	0·1450″	0·1447″	± 0·0016″	**20 Nov. 1885. Alcohol. At 4.30. 504 c.cm.** An effect was clearly perceived at 4.45 and the *b*-series was recognised as slower; there was also some headache. During the *c*-series the headache was more severe and we worked with open doors. Just before the *d*-series A had assisted in another experiment undertaken without my knowledge and requiring continuous mental exertion. This circumstance and the growing severity of his headache led me to reject this series altogether.
III *b*	4.50	50	0·1596″	0·1599″	± 0·0015″	
III *c*	5.20	38	0·1617″	0·1611″	± 0·0027″	
III *d*	6.15	31	[0·2027″]	[0·2028″]	[± 0·0059″]	
IV *a*	4.25	59	0·1429″	0·1423″	± 0·0009″	**3 Dec. 1885. Alcohol. At 4.45. 336 c.cm.** The effect seems to manifest itself in slowing the reaction time and in increasing the probable error, but these changes were not evident to A.
IV *b*	5.15	52	0·1581″	0·1578″	± 0·0028″	
IV *c*	5.47	53	0·1582″	0·1584″	± 0·0021″	
V *a*	4.20	59	0·1670″	0·1657″	± 0·0018″	**10 Dec. 1885. Alcohol. At 4 48. 336 c.cm.** The *b*-series appeared to A to be quicker.
V *b*	4.52	75	0·1579″	0·1580″	± 0·0015″	
V *c*	5.25	60	0·1650″	0·1647″	± 0·0013″	
V *d*	5.50	65	0·1681″	0·1681″	± 0·0015″	
VI *a*	4.07	73	0·1526″	0·1528″	± 0·0010″	**23 Dec. 1885. Normal.** The quick pulls having been recognised as dangerous, they are now avoided and the number of observations retained increases.
VI *b*	4.45	69	0·1584″	0·1587″	± 0·0014″	
VI *c*	5.30	68	0·1515″	0·1516″	± 0·0011″	
VII *a*	3.40	70	0·1417″	0·1416″	± 0·0012″	**30 Dec. 1885. Alcohol. At 4.10. 260 c.cm.** A complained that the festivities of the season had had a depressing effect upon him. During *b* he felt drowsy and had no exhilaration from the alcohol. The *c*-series seemed to him good and all unpleasant effects of the alcohol had gone. Despite A's subjective view the day's work is on the whole the best of any !!
VII *b*	4.20	68	0·1412″	0·1415″	± 0·0011″	
VII *c*	4.50	67	0·1474″	0·1467″	± 0·0008″	

Table I. (*continued*).

Number of series	Time	No. of observations (corrected)	Absolute average	Grouped average	Probable error	Remarks
VIII *a*	2.17	69	0·1484″	0·1482″	± 0·0012″	**25 Jan. 1886. Normal.**
VIII *b*	5.15	72	0·1475″	0·1476″	± 0·0013″	This is the first of a number of experiments with a longer interval. A thought the replies on the whole less good and less trustworthy than usual. Neither the averages nor the errors confirm this idea.
IX *a*	1.40	63	0·1547″	0·1550″	± 0·0017″	**8 Feb. 1886. Normal.**
IX *b*	5.0	67	0·1507″	0·1503″	± 0·0013″	Unfortunately A had drunk nearly half a bottle of claret with his lunch shortly before the *a*-series was taken.
IX *c*	5.30	64	0·1564″	0·1564″	± 0·0015″	
X *a*	1.35	64	0·1480″	0·1484″	± 0·0013″	**15 Feb. 1886. Alcohol.**
X *b*	5.10	63	0·1449″	0·1443″	± 0·0009″	**At 4.0. 260 c.cm.**
X *c*	5.40	64	0·1501″	0·1500″	± 0·0012″	During *c* a slight headache.
XI *a*	2.35	69	0·1431″	0·1425″	± 0·0009″	**27 Feb. 1886. Alcohol.**
XI *b*	4.45	61	0·1561″	0·1563″	± 0·0015″	**At 3.30. 260 c.cm.**
XI *c*	5.20	64	0·1600″	0·1594″	± 0·0013″	Here as in all these experiments with such long intervals A drank the alcohol while away from the laboratory. The day was very cold and windy.
XII *a*	2.35	64	0·1439″	0·1434″	± 0·0010″	**22 March 1886. Alcohol.**
XII *b*	5.15	61	0·1540″	0·1547″	± 0·0015″	**At 4.5. 260 c.cm.**
XII *c*	5.45	69	0·1503″	0·1503″	± 0·0011″	During *b* A felt "some effect of alcohol; not as drowsy as half an hour ago".
XIII *a*	2.45	68	0·1464″	0·1467″	± 0·0010″	
XIII *b*	5.32	70	0·1493″	0·1490″	± 0·0011″	**24 March 1886. Alcohol.**
XIII *c*	6.05	79	0·1509″	0·1510″	± 0·0011″	**At 4.0. 260 c.cm.**
XIV *a*	2.30	67	0·1542″	0·1544″	± 0·0017″	**19 May 1886. Normal.**
XIV *b*	5.18	66	0·1630″	0·1632″	± 0·0020″	The record is of doubtful value, as A had not been well for some days.
XV *a*	2.20	67	0·1601″	0·1601″	± 0·0018″	**22 May 1886. Normal.**
XV *b*	5.12	65	0·1559″	0·1555″	± 0·0015″	A felt better than than on the 19th, but the error is still rather large. The day was warm. His departure for Europe made it impracticable to continue the experiments under more favourable conditions.
XV *c*	5.45	70	0·1606″	0·1607″	± 0·0014″	

TABLE II.

Experiments with D.

Number of series	Time	No. of observations	Absolute average	Grouped average	Probable error	Remarks
I *a*	11.50	50	0·1661″	0·1655″	± 0·0024″	**2 Dec. 1885. Normal.**
I *b*	12.30	46	0·1641″	0·1633″	± 0·0025″	31 "quick pulls" have been removed. The large probable error may possibly be due to lack of practice although a considerable number of records had been taken on previous days.
I *c*	1.0	33	0·1533″	0·1524″	± 0·0024″	
II *a*	3.45	62	0·1621″	0·1622″	± 0·0022″	
II *b*	4.58	61	0·1589″	0·1579″	± 0·0019″	**5 Dec. 1885. Normal.**
II *c*	6.08	64	0·1534″	0·1536″	± 0·0017″	
III *a*	12.0	70	0·1398″	0·1394″	± 0·0015″	**6 Dec. 1885. Alcohol. At 12.30. 140 c.cm.**
III *b*	12.30	70	0·1514″	0·1515″	± 0·0014″	
III *c*	1.0	42	0·1599″	0·1598″	± 0·0021″	Taken in four portions between the sets of the *b*-series and this *b*-series seemed quick! 28 "quick pulls" have been removed.
III *d*	1.30	42	0·1523″	0·1524″	± 0·0020″	
IV *a*	4.45	77	0·2001″	0·2000″	± 0·0015″	**12 Dec. 1885. Alcohol. At 5.20. 252 c.cm. and again at 5.43.**
IV *b*	5.25	71	0·1985″	0·1990″	± 0·0018″	The very slow normal is not accompanied by a large error. D said next day that he had "felt quite blue" this afternoon. During the *c*-series he was somewhat exhilarated and felt "that he could talk better on almost any subject". During the *d*-series he felt "rather dreamy and heavy" but he was distinctly more talkative; he thought the replies of this series "about normal"; although they are really rather high.
IV *c*	5.53	73	0·1793″	0·1792″	± 0·0016″	
IV *d*	6·25	71	0·1685″	0·1685″	± 0·0016″	
V *a*	5.35	74	0·1677″	0·1674″	± 0·0018″	**14 Dec. 1885. Alcohol. At 5.58. 252 c.cm and again at 6.27.**
V *b*	6.02	75	0·1611″	0·1610″	± 0·0014″	
V *c*	6.32	74	0·1603″	0·1603″	± 0·0011″	D felt better than on the 12th. The effect of the alcohol was distinct (subjectively) but not very marked. The series *c* seemed quicker.
V *d*	7.03	75	0·1688″	0·1687″	± 0·0015″	
VI *a*	5.55	70	0·1813″	0·1811″	± 0·0012″	**16 Dec. 1885. Alcohol. At 6.18. 288 c.cm.**
VI *b*	6.23	74	0·1803″	0·1797″	± 0·0016″	The effect was clearly perceived during *b* which seemed quicker than the preceding normal and than the normals in general. During *c* he thought that the effect might have passed off and the series be slower (!); he felt somewhat dull and tired.
VI *c*	6.52	81	0·1598″	0·1600″	± 0·0012″	

Table II. (*continued*).

Number of series	Time	No. of observations	Absolute average	Grouped average	Probable error	Remarks
VII *a*	4.30	73	0·1852″	0·1853″	± 0·0014″	**18 Dec. 1885. Alcohol. At 5.0. 297 c.cm. and again at 5.30.** During *c* and *d* the replies were made with more vigour and with more noise. One rather quick set in *c* was recognised as quick.
VII *b*	5.05	76	0·1728″	0·1727″	± 0·0013″	
VII *c*	5.35	71	0·1631″	0·1622″	± 0·0017″	
VII *d*	6.0	73	0·1665″	0·1665″	± 0·0013″	
VIII *a*	4.40	72	0·1755″	0·1749″	± 0·0019″	**19 Dec. 1885. Alcohol. 3 doses, 297 c.cm. each, at 5.08, 5.34, and 5.58.** Portions of *b*, *c*, and *d* were correctly judged to be quick but his similar opinion of *e* was quite erroneous. Subjectively the effect did not differ much from that of other experiments. Objectively, the replies were unusually energetic and the pencil was laid down with much noise after making a memorandum.
VIII *b*	5.12	75	0·1589″	0·1589″	± 0·0018″	
VIII *c*	5.36	72	0·1533″	0·1533″	± 0·0016″	
VIII *d*	6.04	73	0·1586″	0·1584″	± 0·0014″	
VIII *e*	6.40	74	0·1697″	0·1697″	± 0·0016″	
IX *a*	5.50	56	0·1526″	0·1523″	± 0·0015″	**21 Dec. 1885. Normal.** D thought it less easy to fix his attention but the record gives no evidence of it; in fact the day's work was especially good. 2 cards with 36 observations had to be rejected on account of a mistake in numbering. They did not differ materially from the others.
IX *b*	6.23	54	0·1554″	0·1554″	± 0·0017″	
IX *c*	6.55	71	0·1526″	0·1528″	± 0·0011″	
X *a*	4.20	58	0·1586″	0·1586″	± 0·0017″	**16 June 1886. Alcohol. At 4.37. 520 c.cm.** D thought his attention more easily fixed in *b* and *c* than in *a*, but this view is not borne out by the probable errors.
X *b*	4.50	59	0·1599″	0·1602″	± 0·0023″	
X *c*	5.37	62	0·1628″	0·1631″	± 0·0024″	
XI *a*	3.57	64	0·1476″	0·1471″	± 0·0012″	**18 June 1886. Alcohol. At 4.12. 520 c.cm.** D appeared less affected by this amount than on the 16th, but he thought both *b* and *c* quick. "Attention good."
XI *b*	4.30	68	0·1551″	0·1554″	± 0·0019″	
XI *c*	5.20	68	0·1596″	0·1592″	± 0·0019″	
XII *a*	4.15	71	0·1555″	0·1560″	± 0·0017″	**19 June 1886. Alcohol. At 4.30. 568 c.cm.** Subjectively and objectively the effect was less marked. D thought the *c*-series "if anything a little quicker than without alcohol."
XII *b*	4.50	74	0·1594″	0·1592″	± 0·0016″	
XII *c*	5.38	74	0·1505″	0·1509″	± 0·0018″	
XIII *a*	4.20	69	0·1574″	0·1575″	± 0·0020″	**21 June 1886. Normal.** "Attention good."
XIII *b*	4.55	69	0·1555″	0·1558″	± 0·0018″	
XIII *c*	5.45	74	0·1544″	0·1539″	± 0·0016″	

TABLE III.

Experiments with W.

Number of series	Time	No. of observations	Absolute average	Grouped average	Probable error	Remarks
I *a*	4.20	55	0·1635″	0·1632″	± 0·0018″	**5 Dec. 1885. Normal.**
I *b*	5.25	52	0·1739″	0·1749″	± 0·0017″	The cause of the large probable error is not quite clear. It is noted that the shock during *c* was feeble.
I *c*	6.30	31	0·1773″	0·1781″	± 0·0034″	
II *a*	3.45	53	0·1706″	0·1710″	± 0·0025″	
II *b*	4.35	58	0·1705″	0·1707″	± 0·0013″	**7 Dec. 1885. Normal.**
II *c*	5.15	54	0·1794″	0·1794″	± 0·0013″	
III *a*	12.0	70	0·1675″	0·1674″	± 0·0009″	**13 Dec. 1885. Normal.**
III *b*	12.55	69	0·1620″	0·1624″	± 0·0008″	
IV *a*	11.45	54	0·1687″	0·1689″	± 0·0017″	**16 Dec. 1886. Normal.**
IV *b*	12.40	57	0·1680″	0·1680″	± 0·0015″	Just after giving a 2 hours course of laboratory instruction. It was not easy to fix the attention.
V *a*	12.15	79	0·1609″	0·1609″	± 0·0010″	**20 Dec. 1885. Alcohol.**
V *b*	12.58	84	0·1675″	0·1675″	± 0·0008″	**At 12.55. 198 c.cm.**
V *c*	1.37	88	0·1646″	0·1641″	± 0·0012″	Some "dreaminess" during *c*.
VI *a*	12.20	34	0·1569″	0·1564″	± 0·0022″	**27 Apr. 1886. Normal.**
VI *b*	1.26	44	0·1682″	0·1683″	± 0·0017″	No apparent reason for the large probable error except possibly want of practice.
VII *a*	11·45	57	0·1593″	0·1594″	± 0·0012″	**28 Apr. 1886. Alcohol.**
VII *b*	12.44	63	0·1772″	0·1773″	± 0·0020″	**At 12.8. 280 c.cm.**
VII *c*	1.30	56	0·1762″	0·1766″	± 0·0015″	At 12.15 distinct exhilaration. During *b* and *c* much difficulty in fixing attention. The replies seemed "hampered" and were recognised as probably slow.
VIII *a*	11.55	62	0·1603″	0·1608″	± 0·0013″	**29 Apr. 1886. Alcohol.**
VIII *b*	12.50	60	0·1674″	0·1676″	± 0·0013″	**At 12.15. 280 c.cm.**
VIII *c*	1.26	63	0·1660″	0·1653″	± 0·0014″	"Not feeling very well." The effect of the alcohol was subjectively distinct. The replies in *b* seem hampered.

Table III. (*continued*).

Number of Series	Time	No. of observations	Absolute average	Grouped average	Probable error	Remarks
IX *a*	10.50	63	0·1650″	0·1653″	± 0·0015″	**30 Apr. 1886. Alcohol.**
IX *b*	11.50	65	0·1631″	0·1628″	± 0·0012″	**At 11.12. 280 c.cm.**
IX *c*	12.50	63	0·1595″	0·1586″	± 0·0010″	Note-book shows as usual an increase of introspectiveness. W thought the replies might be slow.
X *a*	11.30	69	0·1626″	0·1629″	± 0·0015″	**5 May 1886. Alcohol.**
X *b*	12.18	73	0·1686″	0·1689″	± 0·0013″	**At 11.53. 420 c.cm.**
X *c*	12.58	68	0·1635″	0·1637″	± 0·0011″	Very marked subjective effect. W felt sure that the replies in *b* must be slow but was equally sure of those in *c*.
XI *a*	11.22	58	0·1545″	0·1541″	± 0·0009″	**6 May 1886. Alcohol.**
XI *b*	12.18	59	0·1606″	0·1604″	± 0·0011″	**11.40. 420 c.cm.**
XI *c*	1.7	56	0·1580″	0·1573″	± 0·0012″	Subjectively less but objectively more affected than on the 6th. Judgment warped as above.
XII *a*	11.48	63	0·1547″	0·1547″	± 0·0010″	**7 May 1886. Alcohol.**
XII *b*	1.28	61	0·1664″	0·1663″	± 0·0013″	**At 12.12. 430.5 c.cm.**
XIII	12.0	55	0·1559″	0·1561″	± 0·0009″	**17 May 1886. Normal.** Unexpectedly prevented from taking a later record.
XIV *a*	11.45	55	0·1574″	0·1569″	± 0·0008″	
XIV *b*	12.40	60	0·1679″	0·1681″	± 0·0014″	**18 May 1886. Normal.**
XIV *c*	1.20	67	0·1625″	0·1628″	± 0·0010″	The *b*-series seemed a "very attentiv record"!

An examination of these records shows that the limits both of the "normals of the day" and of the changes in the alcohol days are much less for A and W than for D. The "normals of the day" lie for A between 0·1417″ and 0·1670″, for W between 0·1545″ and 0·1706″, while those for D range from 0·1398″ to 0·2001″, all exclusive of the probable error. For this remarkable variation in D's replies no cause could be found except in a single instance, that of "feeling blue" when the very slow normal, 0·2001″ (in IV *a*), was obtained. For the sake of comparison these normals are given in Table IV. in the order of their size. This table shows that normals of the day lie fairly well together for W and for A, while those of D are so spread out as to include both extremes. The table also shows us that the slow averages are not due to any bad-

ness of the replies as indicated in the probable error; and an examination of the order of the observations does not make it evident that practice improved the replies very materially except possibly in the case of W. For the purpose of making such comparison easier Table V. has been prepared, giving the normals of the day chronologically. The poorness of A's records in May, 1886, both as to averages and as seen in the probable error may perhaps be attributed to the condition of his health at that time as already mentioned.

TABLE IV.

"NORMALS OF THE DAY" ARRANGED ACCORDING TO THEIR SIZE, TOGETHER WITH THE NUMBER OF OBSERVATIONS AND THE CORRESPONDING PROBABLE ERROR (IN TEN-THOUSANDTHS OF A SECOND).

A.				D.				W.			
		No. of observations.	Probable error.			No.	Error.			No.	Error.
				IV	0·2001″	77	±15				
				VII	0·1852″	73	±14				
				VI	0·1813″	70	±12				
				VIII	0·1755″	72	±19				
								II	0·1706″	53	±25
								IV	0·1687″	54	±17
V	0·1670″	59	±18	V	0·1677″	74	±18	III	0·1675″	70	± 9
				I	0·1661″	50	±24	IX	0·1650″	63	±15
								I	0·1635″	55	±18
				II	0·1621″	62	±22	X	0·1626″	69	±15
								V	0·1609″	79	±10
XV	0·1601″	67	±18					VIII	0·1603″	62	±13
				X	0·1586″	58	±17	VII	0·1593″	57	±12
				XIII	0·1574″	69	±20	XIV	0·1574″	55	± 8
								VI	0·1569″	34	±22
				XII	0·1555″	71	±17	XIII	0·1559″	55	± 9
IX	0·1547″	63	±17					XII	0·1547″	63	±10
XIV	0·1542″	67	±17					XI	0·1545″	58	± 9
VI	0·1526″	73	±10	IX	0·1526″	56	±15				
VIII	0·1484″	69	±12								
II	0·1483″	45	±14								
X	0·1480″	64	±13	XI	0·1476″	64	±12				
XIII	0·1464″	68	±10								
I	0·1456″	32	±25								
III	0·1450″	43	±16								
XII	0·1439″	64	±10								
XI	0·1431″	69	± 9								
IV	0·1429″	59	± 9								
VII	0·1417″	70	±12								
				III	0·1398″	70	±15				

TABLE V.

"NORMALS OF THE DAY" ARRANGED *CHRONOLOGICALLY*, TOGETHER WITH THE NUMBER OF THE OBSERVATIONS AND THE PROBABLE ERROR (IN TEN-THOUSANDTHS OF A SECOND).

	A.			D.			W.		
		No. of observations.	Probable error.		No.	Error.		No.	Error.
1885									
15 Nov.	0·1456″	32	±25						
17 ,,	0·1483″	45	±14						
20 ,,	0·1450″	43	±16						
2 Dec.				0·1661″	50	±24			
3 ,,	0·1429″	59	± 9						
5 ,,				0·1621″	62	±22	0·1635″	55	±18
6 ,,				0·1398″	70	±15			
7 ,,							0·1706″	53	±25
10 ,,	0·1670″	59	±18						
12 ,,				0·2001″	77	±15			
13 ,,							0·1675″	70	± 9
14 ,,				0·1677″	74	±18			
16 ,,				0·1813″	70	±12	0·1687″	54	±17
18 ,,				0·1852″	73	±14			
19 ,,				0·1755″	72	±19			
20 ,,							0·1609″	79	±10
21 ,,				0·1526″	56	±15			
23 ,,	0·1526″	73	±10						
30 ,,	0·1417″	70	±12						
1886									
25 Jan.	0·1484″	69	±12						
8 Feb.	*0·1547″	63	±17						
15 ,,	0·1480″	64	±13						
27 ,,	0·1431″	69	± 9						
22 March	0·1439″	64	±10						
24 ,,	0·1464″	68	±10						
27 April							0·1569″	34	±22
28 ,,							0·1593″	57	±12
29 ,,							0·1603″	62	±13
30 ,,							0·1650″	63	±15
5 May							0·1626″	69	±15
6 ,,							0·1545″	58	± 9
7 ,,							0·1547″	63	±10
17 ,,							0·1559″	55	± 9
18 ,,							0·1574″	55	± 8
19 ,,	0·1542″	67	±17						
22 ,,	0·1601″	67	±18						
16 June				0·1586″	58	±17			
18 ,,				0·1476″	64	±12			
19 ,,				0·1555″	71	±17			
21 ,,				0·1574″	69	±20			

* Had taken some claret with his lunch.

The probable error shows some lessening with practice. But the number of observations in the first 3 records is perhaps too small for comparison.

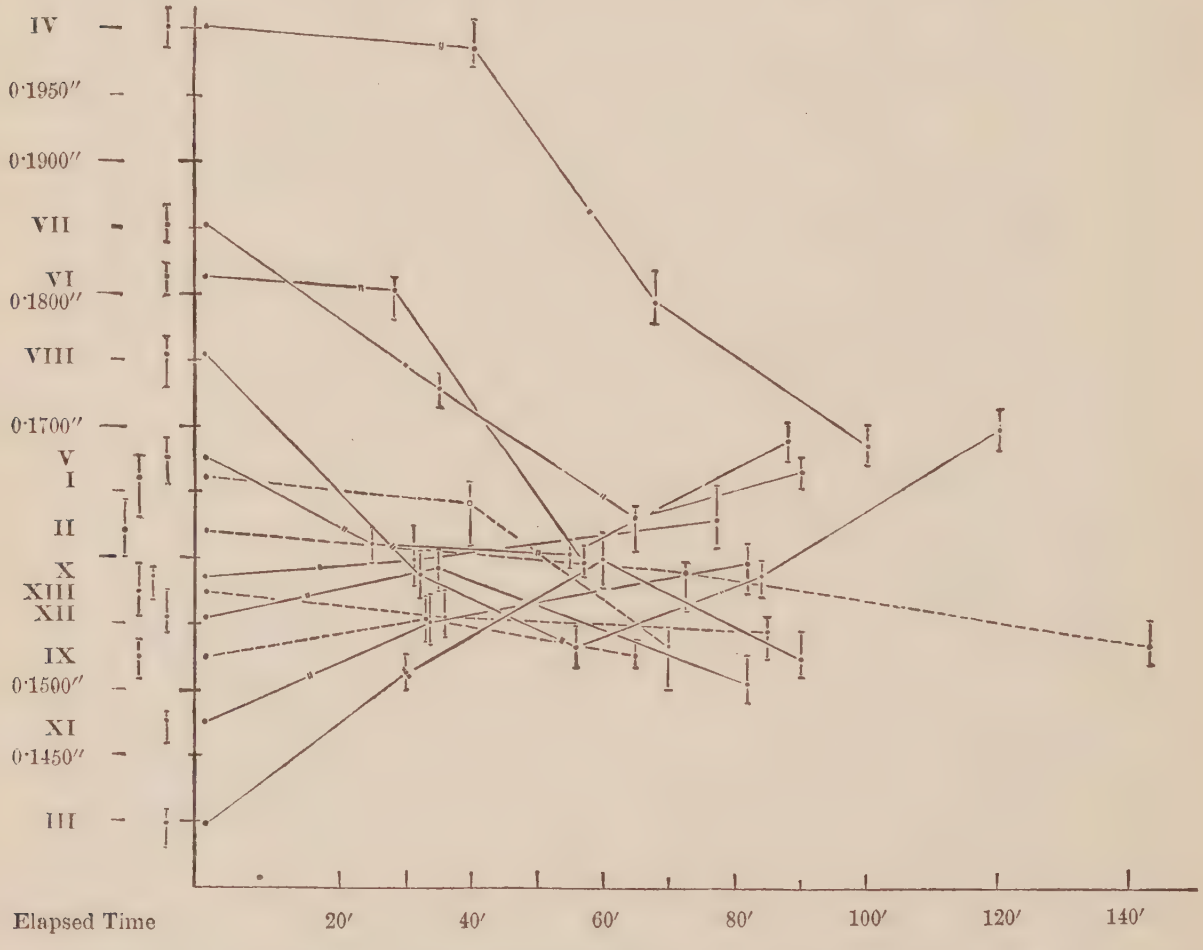
There is no constancy in the improvement after the 5th of Dec. as shown in the probable error.

There is some improvement with practice both in the averages and in the probable errors. Obviously the small number of observations on 27th April forbids a comparison of probable errors.

I have drawn many curves for my own inspection which give not only the position of the normals but also the course of the experiments both with alcohol and without it. As such curves show very little that is interesting concerning the reactions of A and of W, I only print those showing the results obtained in the experiments with D, as seen in Fig. 4. In this diagram the ordinate gives the value of the absolute average in ten-thousandths of a second, the time from the beginning of the day's work (or the "elapsed time in the experiment" as I shall call it) being indicated in minutes on the abscissa. The time at which the

Fig. 4.

Averages in the experiments with D arranged according to the size of the normals of the day and with reference to the elapsed time of each experiment.



In this diagram the time when alcohol was taken is noted by breaks in the otherwise full lines of the alcohol experiments. Where more than one dose was used each is noted in its place. In curve III it will be observed that the alcohol mark and the second "*b*" average fall together, i.e. no time had elapsed, the alcohol being drunk at intervals during the experiment. At the left of the perpendicular line the probable errors of the normals of the day are shown graphically to exhibit such overlapping as occurs. The number belonging to each experiment as detailed in Table II. stands at the left among the numerals which mark the value of the observations.

alcohol was taken is shown by the dotted break in the otherwise solid lines of the alcohol curves. The "normal" curves are shown as broken lines, the beginning of each curve being of course the "normal for the day". The perpendicular line drawn through each dot (representing an "absolute average") marks the extent of the probable error.

The alteration which occurs in the reaction time during the course of an experiment may be represented graphically in another way. Evidently it is of importance to know not so much the value of the absolute averages with reference to those of other days, but rather to compare those on one and the same day in order to learn the amount of change which occurs either with alcohol or normally. In other words, it appears more profitable to consider the "change" which any dose of alcohol may have produced than to know the exact value of any average itself. If we take the "normal of the day" as the starting point for the day's work (as is obviously the fairest way of doing) we may call the difference between that average and any other one average on the same day the "change" and mark it + or − according as the reaction time has grown larger or smaller. This "change" will have a "range" of its own (as already explained above) since it really depends upon two "probable errors" as well as upon two "absolute averages" for its amount. That is to say, if we place the normal observation for each day upon a zero line we can group the changes above the line (for a slowing) or below it (for a quickening). If in addition to this we let the zero line be an abscissa on which we mark the time during which alcohol has been at work we shall have a convenient and compendious view of the alcohol effect. The diagrams in Figures 5, 6, and 7 give then a graphic statement of the relation of all the later observations to their individual normals. The position of each dot indicates the character and amount of the difference between the two absolute averages in question the range of such change being the length of the perpendicular line drawn through the dot. Wherever the range line crosses the zero line we must infer that we are not justified in supposing that the second reaction time under consideration is really greater or less than its normal, it may or may not be so. For the sake of comparison the changes of the normal days are given as well as those which may be attributed to alcohol. These are printed as broken lines and of course can be arranged only according to the time which has elapsed since the experiment began. Each "normal" change is accordingly placed as near as practicable to the "alcohol" change which has the same "elapsed time." Below each dot will be found the data

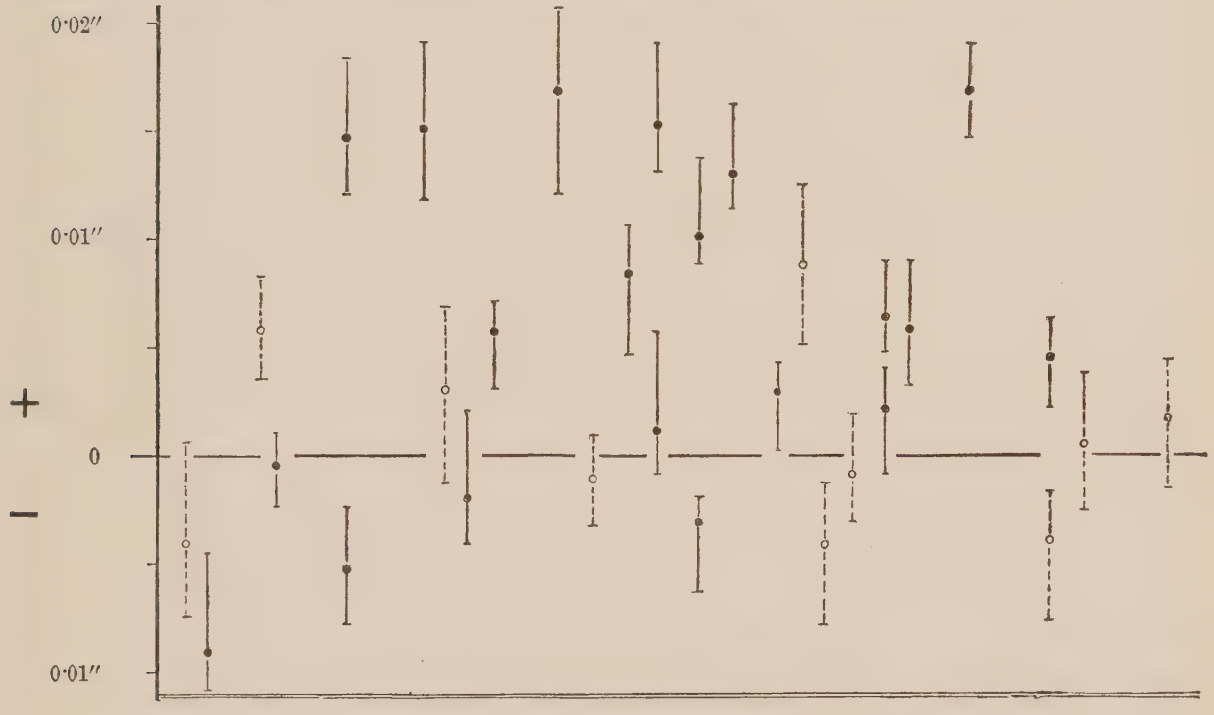
necessary for such comparisons as the reader may wish to make. The Roman numeral indicates the number of the series whose comparison with its normal has resulted in the change recorded. The amount of alcohol is noted in cubic centimetres of a ten per cent. solution. The full figures of these curves will also be found in Table VI., where they are arranged so as to permit a comparison of the changes for A, D, and W, still more readily. The corresponding changes of the "normals" are noted in Table VII. To be more explicit, Fig. 5 for example contains all the changes found in the observations made on A as recorded above in Table I. The first change in order is shown as a broken line, it belongs therefore to a day's work where no alcohol was taken; just below it in the rows belonging to "normal" we find I *b* and 30′, meaning that a series of records in the first experiment with A taken thirty minutes after the experiment began (or the *a*-series was taken) shows the change recorded. This change is –, or the time was quickened, and not far from 0·005″ in amount; but the broken line which marks the "range" crosses the zero line, it is therefore an open question whether the reaction time have been really quickened or not. If we wish the exact figures represented by this line we shall find them in Table VII. in A's column and in the line belong to an "elapsed time" of 30—33′; the change is then − 0·0042″ with a "range" from + 0·0006″ to − 0·0076″.

Next to this broken line is a plain black line; it is therefore a change associated with alcohol. Corresponding to it in the "alcohol" rows below are the figures V *b*, 4′, 336; it is then a change observed in the second (or *b*-) series of the fifth experiment with A and related to the influence of 336 c.cm. of 10 % alcohol drunk four minutes before this series was taken. Referring to Table VI. we find the exact change to be − 0·0091″ with a range from − 0·0045″ to − 0·0109″, it is therefore an unequivocal quickening. Next in the diagram comes a "normal" change and the memoranda below show that it belongs to the *b*-series of the sixth experiment and was observed 38 minutes after the experiment began. Then follows a slight and dubious quickening belonging to the *b*-series of the seventh experiment and possibly associated with the 260 c.cm. of alcohol consumed ten minutes before. We then find two alcohol changes in one line because in both the time during which alcohol has been in the body is twenty minutes; examining the figures below we see that the upper change (or slowing) belongs to III *b* and has been brought about by 504 c.cm. alcohol, while the lower one (quickening) belongs to II *b*, in which case 224 c.cm. of alcohol had been taken.

In this way the three diagrams are to be read. The elapsed time for the normals and the periods of time during which the alcohol has been acting are not fully detailed but so placed as to show the general relations of the observations and, as I have already mentioned, the elapsed time in the alcohol experiments (i.e. the absolute time of the experiment without regard to the duration of any alcohol effect) is usually very nearly the same as that of the normal experiments placed near them in the diagram.

FIG. 5.

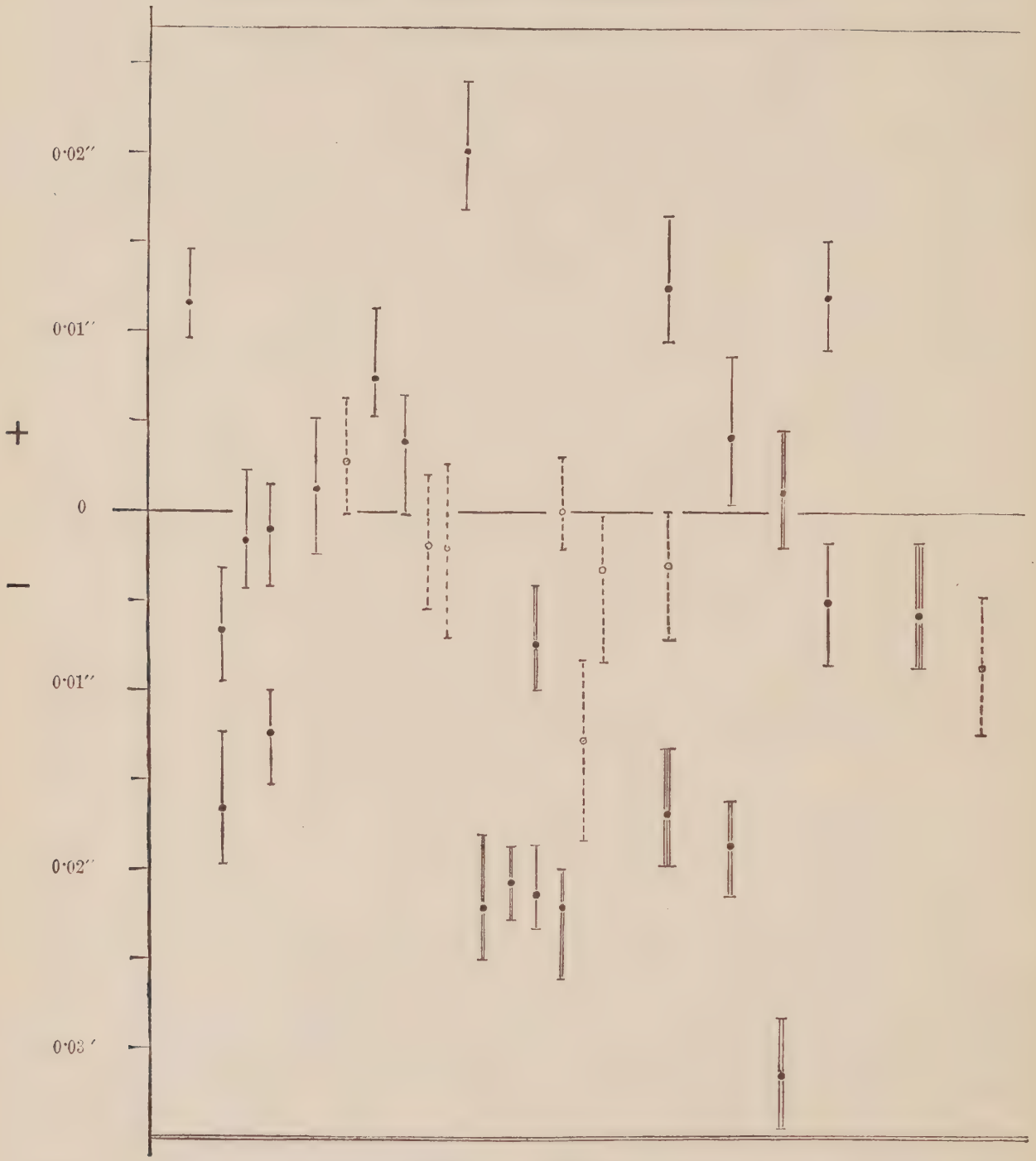
"CHANGES" AND "RANGES" OCCURRING IN THE EXPERIMENTS WITH A.



| | | |

FIG. 6.

"CHANGES" AND "RANGES" IN EXPERIMENTS WITH D.

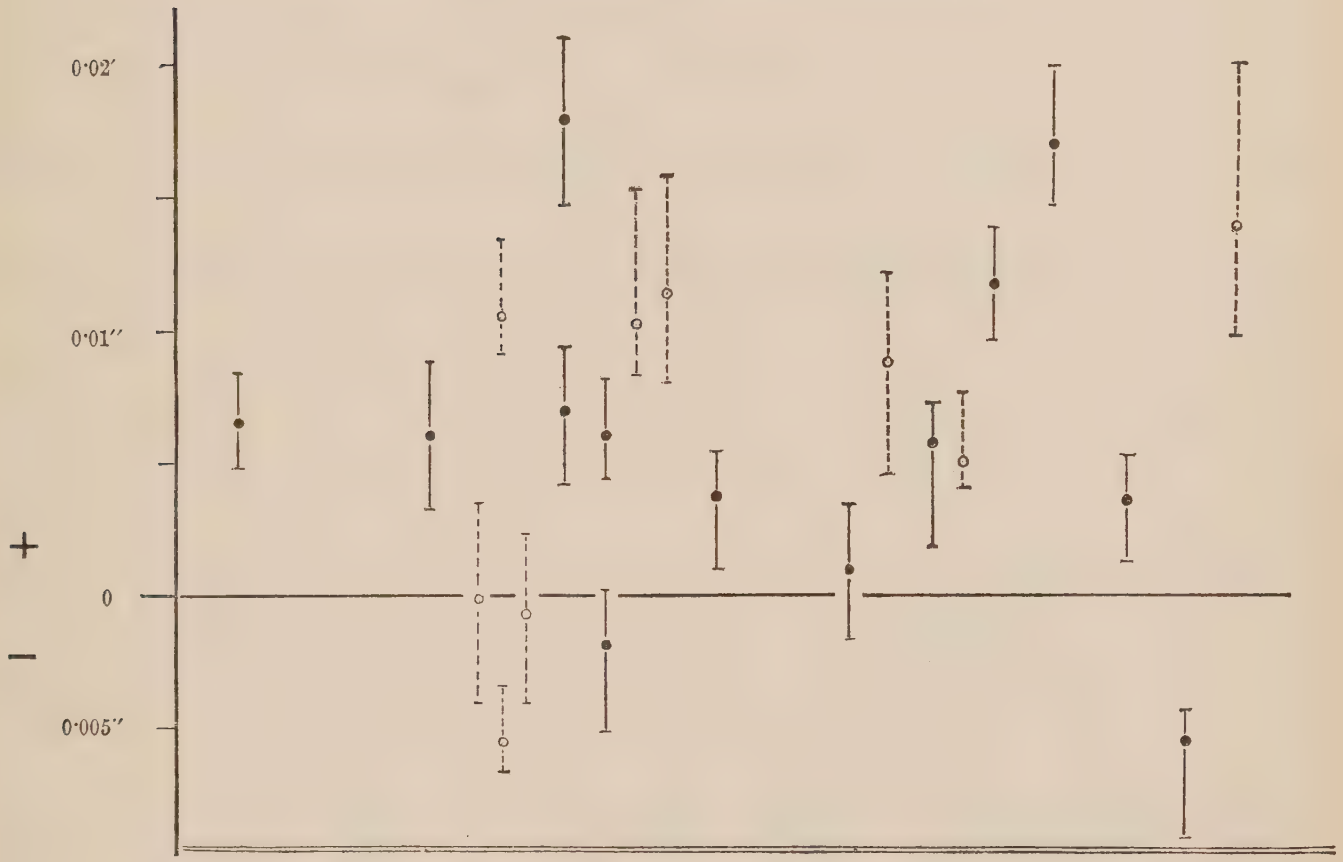


| | | |

taken, the single black line as in the other diagrams belonging to one single dose. The time noted below a double or triple dose only tells how many minutes have passed since the *first* dose was taken and the amount noted is the quantity taken in *each* dose. The time which has passed since the second or third dose may be calculated from Table II. and is also noted in connection with Fig. 8, below. By x' is meant that the number of minutes is not exactly known, the alcohol having been taken at intervals while a record was being made.

FIG. 7.

"CHANGES" AND "RANGES" IN EXPERIMENTS WITH W.



Normal				XIV^b II^{b}III^{b}IV^b		I^b VI^b			II^c	XIV^c					I^c
Elapsed time				50′		65′			90′						130′
Alcohol	V^b		X^b	VII^b XI^b VIII^{b}IX^b			V^c		X^c	VIII^c	XII^b	VII^c	XI^c	IX^c	
Time	3′		25′	35′			42′		65′			82′		98′	
Amount of alcohol	198		420	280	420 280		198		420	280	430·5	280	420	280	

TABLE VI. CHANGE AND RANGE OF THE ALCOHOL SERIES, ARRANGED FOR TIME AFTER THE (FIRST) DOSE OF ALCOHOL. THE FIGURES OF THE "RANGE" INDICATE TEN-THOUSANDTHS OF A SECOND.

	A.			D.			W.		
Time.	Alcohol.	Change.	Range.	Alcohol.	Change.	Range.	Alcohol.	Change.	Range.
x'				140	+0·0116″	+146 +96			
3–4′	336	−0·0091″	−45 −109	252	−0·0066″	−32 −96	198	+0·0066″	+84 +48
4′				297	−0·0166″	−123 −197			
5′				252	−0·0016″	+23 −43 } =0			
				288	−0·0010″	+15 −42 } =0			
				297	−0·0124″	−100 −153			
10′	260	−0·0005″	+22 −24 } =0						
13′				520	+0·0013″	+57 −24 } =0			
18′				520	+0·0075″	+114 +53			
20′	224	−0·0053″	−24 −78	568	+0·0039″	+65 −1 } =0			
	504	+0·0147″	+183 +121						
25′				140	+0·0201″	+240 +168	420	+0·0060″	+88 +32
28′				2× 297	* * −0·0222″	−181 −251			
30′	336	+0·0152″	+192 +118						
33′				2× 252	* * −0·0208″	−188 −229			
34′				288	−0·0215″	−187 −234			
34–5′				2× 252	* * −0·0074″	−41 −100	280	+0·0071″	+94 +42
35–6′				2× 297	−0·0221″	−200 −262	280	+0·0179″	+211 +147
37–8′	336	−0·0020″	+21 −41 } =0				280	−0·0019″	+2 −52 } =0
38′							420	+0·0061″	+82 +44
			+71						+51

55′					140	+0·0125″	+105			
56′					3 × 297	*** −0·0169″	+95 −132 −198			
60′	224	+0·0084″	+107 +47		520 2 × 297	+0·0042″ ** −0·0187″	+87 } =? +4 } −162 −215			
62′	336	+0·0153″	+191 +132							
65′	336	+0·0011″	+57 } =0 −9 }		2 × 252 2 × 252	** +0·0011″ ** −0·0316″	+46 } =0 −20 } −284 −346	420	+0·0009″	+34 } =0 −18 }
68′					520	+0·0120″	+152 +91			
70′	260	−0·0031″	−19 −63		568	−0·0050″	−17 −86	280	+0·0057″	+72 +18
75′	260	+0·0101″	+137 +88					430·5	+0 0117″	+139 +95
82′	260	+0·0130″	+162 +114					280	+0·0169″	+199 +146
87′								420	+0·0035″	+52 +12
92′	260	+0·0029″	+43 } =? +2 }		3 × 297	*** −0·0058″	−17 −87			
98′								280	−0·0055″	−43 −92
100′	260	+0·0021″	+41 } =0 −9 }							
	260	+0·0064″	+90 +48							
110′	224	+0·0058″	+90 +32							
110′	260	+0·0169″	+191 +147							
125′	260	+0·0045″	+63 +22							

or =0 means that the range line nearly or quite cuts the zero line. ** two doses. *** three doses of alcoh

TABLE VII.

CHANGE AND RANGE OF THE NORMALS ARRANGED FOR TIME FROM BEGINNING OF EXPERIMENT. THE FIGURES OF THE RANGE INDICATE TEN-THOUSANDTHS OF A SECOND.

	A.		D.		W.	
Time.	Change.	Range.	Change.	Range.	Change.	Range.
30–33′	−0·0042″	+6, −76 } = 0	+0·0028″	+63, −1 } = 0		
35′			−0·0019″	+21, −55 } = 0		
38–40′	+0·0058″	+83, +35	−0·0020″	+27, −71 } = 0		
50′					−0·0001″	+35, −41 } = 0
55′					−0·0055″	−33, −66
					−0·0007″	+23, −41 } = 0
					+0·0105″	+134, +91
60′	+0·0030″	+69, −13 } = 0				
65′			±0·0000″	+31, −21 } = 0	+0·0104″	+152, +83
66′					+0·0113″	+157, +80
70′			−0·0128″	−83, −179		
73′			−0·0032″	−2, −84 } = ?		
83–85′	−0·0011″	+9, −33 } = 0	−0·0030″	−72, ±00 } = 0		
90′					+0·0088″	+122, +46
95′					+0·0051″	+77, +41
130′					+0·0138″	+200, +97
143′			−0·0087″	−47, −125		
168′	+0·0088″	+125, +51				
172′	−0·0042″	−13, −79				
178′	−0·0009″	+19, −31 } = 0				
200′	*−0·0040″	−17, −77				
205′	+0·0005″	+38, −26 } = 0				
230′	*+0·0017″	+44, −16 } = 0				

* After claret!

An examination of these diagrams and of the corresponding tabulation will make it evident that the changes are more frequent where alcohol is taken than where none has been drunk. This is especially evident if we compare the cases where the range line crosses the zero line or comes so near as to very much lessen the trustworthiness of the change. The percentage of cases where this occurs will be found to be

For	A.	D.	W.
In "Normal" series	60 %	75 %	22 %
In "Alcohol" „	25 %	25 %	15 %

In other words, the range reduces the change to zero much less frequently where alcohol has been at work[10]. This effect is not so marked for W as for the others. It is however possible that his normals are less trustworthy—perhaps on account of insufficient practice. The size of the probable errors suggests such an explanation; still the proportions for those cases where no such objection can be urged is about the same.

A comparison of the frequency of increase and decrease (that is of all the cases where the change is not reduced to zero) is also interesting. The following little table gives the percentage:

	A.		D.		W.	
	+	−	+	−	+	−
Normally	20 %	20 %	0 %	25 %	67 %	11 %
Alcohol	60 %	15 %	21 %	54 %	77 %	8 %

[10] This is even more marked if we remove certain comparisons where the variation in the number of observations seems to render the comparison less fair (as in D, I *c*, III *c*, III *d*, IX *c*; and W, I *c*) We should then find the change equal to zero,

For	A.	D.	W.
Normally in	60 %	83 %	25 %
Under alcohol in	25 %	27 %	15 %

Evidently then A's records are much more frequently slowed when alcohol is taken than otherwise, while those of D are much oftener quickened. Here again W makes a much less definite showing than the others, the slowing effect being less well marked.

Another way of looking at the changes leads to a similar result. If we add together all the absolute changes which are marked + and take the average (without reference to the number of single records) and also do the same with the minus changes or quickenings, the average change will prove to be:

A.		D.		W.	
Normally.	Alcohol.	Normally.[11]	Alcohol.	Normally.	Alcohol.
+ 0·0040″	0·0093″	0·0014″	0·0082″	0·0100″	0·0078″
− 0·0029″	0·0040″	0·0045″	0·0140″	0·0021″	0·0037″

Here again alcohol has a marked influence on the records of A and D, but its influence on those of W is partly in the opposite direction, nor does this variation appear to be caused by the character of his normals[12].

Neither the amount of alcohol nor the time which has passed since taking it seems to have any distinct or regular influence on the change produced[13]. Only the early changes for A seem in agreement with the statement of others that a primary quickening may be expected and that the later effect will be a slowing, but neither the later changes in A's records nor the variations for D and W corroborate this opinion. Although I have compared the records from many points of view (with reference to amount of alcohol, to the time of day, etc.) the curves have never resulted in the establishment of any general relations of the least definiteness.

The remarkable variation in the normal reaction times of D as well as the frequent quickening by alcohol in his case suggests a comparison between the effect of the alcohol and the size of the normal of the day.

[11] The ±0·0″ in IX *c* being reckoned twice.

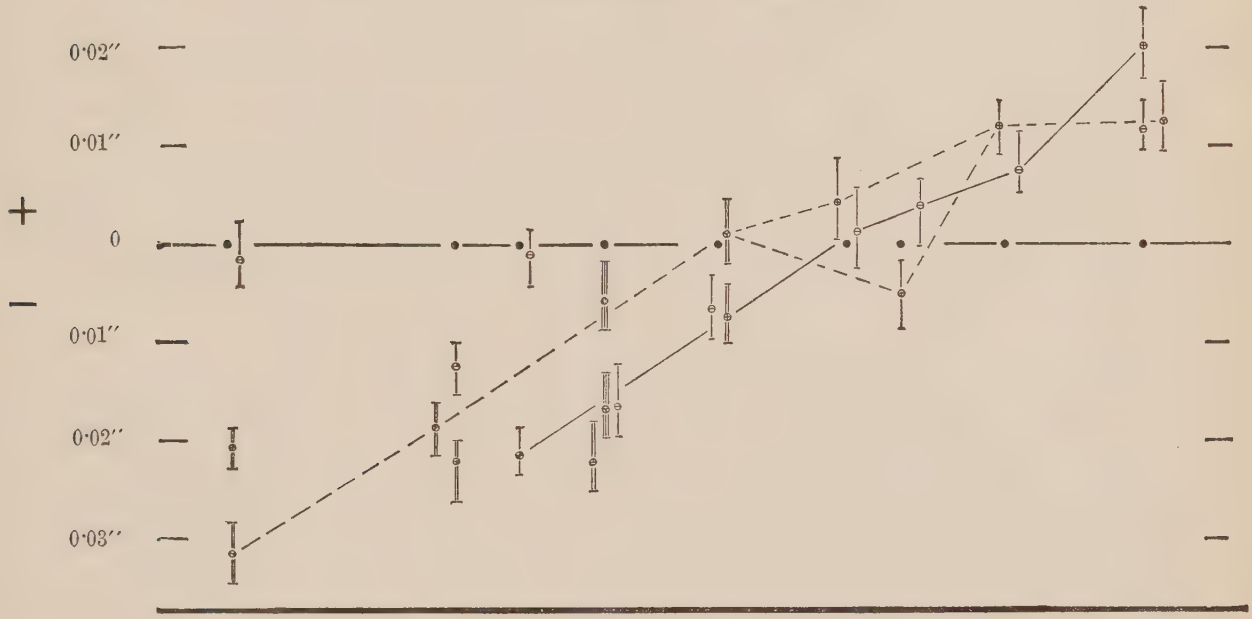
[12] The omission of I and II for W, where lack of practice might be suspected, does not materially alter the above table.

[13] It will be observed that very few of A's series show an unequivocally more marked effect for the later times (after intervals of 30′—50′). And among W's records there is no case where the later effect of alcohol is positively more marked than the earlier effect on the same day, the interval being generally about 40′.

This is also suggested by the peculiarity in the grouping which manifests itself in the diagram of Fig. 4. If we arrange these "ante-alcoholic" normals along our zero line in order and make the intervals proportionate to the size of the normals, we may draw the changes above or below the line as we have done before. The Diagram in Fig. 8 has been prepared in this way and gives all the changes (and their ranges) belonging to the ante-alcoholic normals near which they stand. It will be seen that the changes which occur in the first few minutes after taking alcohol are exceedingly irregular, and this is owing perhaps to irregularities in

FIG. 8.

"CHANGES" AND "RANGES" OF THE ALCOHOL EXPERIMENTS WITH D ARRANGED ACCORDING TO THE SIZE OF THE NORMALS OF THE DAY.



Value of normal		0·2001″	0·1852″	—	0·1755″	0·1677″	—	0·1555″	—	0·1398″
Running number		IV	VII	VI	VIII	V	X	XII	XI	III
Time since the alcohol was taken in	*b*	5′	5′	5′	4′	4′	13′	20′	18′	*x*′
	c	33+10′	35+5′	34′	28+2′	34+5′	60′	68′	68′	30 − *x*′
	d	65+42′	60+30′	—	56+30 +6′	65+36′	—	—	—	60 − *x*′
	e	—	—	—	92+66 +42′	—	—	—	—	—

In the above table the range line is a simple line or otherwise according as the alcohol was given in one, two, or three doses. The position of the "normals of the day" (or the *a*-series) is shown by the solid dots in the zero line. The absolute changes occurring in the *b*-series are marked by open dots with a single crossing line, those for the *c*-series by two crossing lines, and in *d*-series the dot has three radial lines. The time noted below the diagram shows how long the alcohol has been in the body. Thus, in the various series belonging to the day marked VIII, the *b*-series was taken 4′ after the alcohol had been drunk, the *c*-series 28 minutes after the first dose of alcohol and 2 minutes after the second; when the *d*-series began 56 and 30 minutes had elapsed since the first and second doses respectively, and 6 minutes since the consumption of the third.

the absorption. But if we draw a line through those dots which show the effect where the time is such that a distinct result from one dose may be expected (i.e. after 30-x', 18′, 20′, 13′ and 34′ of III *c*, XI *b*, XII *b*, X *b*, and VI *c* respectively) we have *almost a straight line.* This line it will be seen also lies near the changes probably produced by a single dose in 34′, 28′ and 35′ (V *c*, VIII *c*, and VII *c*) where the second dose has perhaps not had time to cause much further change. I have also dotted in a line to connect the later effects, but the result is not striking. It would seem then that where the normal reaction times vary greatly the effect of alcohol is to steady them, raising those that are low and quickening those that are slow. It was quite accidental that I happened to select just these variable days for alcohol work, since I knew nothing of the value of the averages (except perhaps in one or two cases) for many weeks after the records were taken. As neither A nor W shows any such great variation in the normals a similar comparison of the alcohol effect upon them is impossible, and would be complicated by much overlapping of the normals, while among D's "ante-alcoholic normals" only X *a* and XII *a* overlap one another by reason of the size of their probable errors.

The experiments with D are the only ones where the influence of a second or third dose of alcohol was tested, and the effect is complicated by the fact that these experiments happened to have high normals. It will be seen that a second dose does not constantly quicken even where the normal is high, and that the third dose caused a relative slowing.

The only other points of interest connected with this research would seem to be the influence of alcohol on the probable error and also upon the rejected observations. Table VIII. gives a full list of the probable errors. Obviously, if we wish to compare them in order to study the amount of change in the error from one series to another, it will be unfair to institute a comparison where the number of observations made in the two series varies greatly. To facilitate such an examination the number of reaction times corresponding to each probable error is placed above it. Thus while we may compare the series of D II it will evidently not do to compare D I *a* with D I *c* nor D III *a* with the later D III *c* and III *d*. The alcohol series under "*a*," it will be remembered, belong to "ante-alcoholic" normals. Examined then in this sense (i.e. omitting: for A, V *b*, XIII *c*, II *d*; for D, I *c*, IX *c*, III *c*, III *d*, VI *c*; for W, I *c*, VI *b*, XIV *c*, V *c*), the later observations of different days compared

with their own "normals of the day" show an increase or decrease of the probable error as follows:

	Normally.		After Alcohol.	
	Increase in	Decrease in	Increase in	Decrease in
For A.	40 %	60 %	51 %	49 %
„ D.	33 %	67 %	48 %	52 %
„ W.	17 %	83 %	54 %	46 %

It appears then that the normal series more frequently show an improvement in the quality of the replies given in the course of the day's work than do those series which are presumably influenced by alcohol; in other words, the latter series seem to be rather less trustworthy than the former. If these changes in the normal observations be really due to practice it is still true, as already mentioned, that a similar change is not clearly manifest when the normals of the day are compared chronologically.

But if we examine the probable errors with reference to the amount rather than the frequency of change in them in the course of each day, it will be seen that the trustworthiness of A's records is really much more disturbed than that of D or of W; indeed the effect on W's probable errors, and to a less extent on those of D, is such that their alcohol replies seem on the whole to be better balanced—to be steadied. Obviously the number of experiments is too small and the conditions are too varied to give such comparisons any positive value. Table VIII. gives also no satisfactory evidence that the probable errors vary in proportion to the amount of alcohol nor to the duration of its action in the various experiments.

Of the rejected records, i.e. of those which were called "bad" or "slow" although in reality fairly good, a distinctly larger proportion occurred in "normal" series; of those called "good", but which were really so far away from the rest of their groups as to lead to their rejection, a larger proportion belonged to "normals" for D and W, while A made this mistake rather more frequently in his alcohol series, and the same is true of his error in giving absurdly quick replies in a few cases. In brief, alcohol apparently did not particularly warp the judgment of D and W and only very slightly that of A as shown in this way. No more marked effect is evident in the judgments

TABLE VIII.

Tabulation of the Probable Errors together with the number of observations on which they are based. The error in ten-thousandths of a second.

I. Normal Series.

	A.					D.						W.		
	a	*b*	*c*	*d*		*a*	*b*	*c*	*d*	*e*		*a*	*b*	*c*
I	32 ±25	36 ±16	39 ±17		I	50 ±24	46 ±25	33 ±24			I	55 ±18	52 ±17	31 ±34
VI	73 ±10	69 ±14	68 ±11		II	62 ±22	61 ±19	64 ±17			II	53 ±25	58 ±13	54 ±13
IX	63 ±17	67 ±13	64 ±15		IX	56 ±15	54 ±17	71 ±11			III	70 ±9	69 ±8	
VIII	69 ±12	72 ±13			XIII	69 ±20	69 ±18	74 ±16			IV	54 ±17	57 ±15	
XIV	67 ±17	66 ±20									VI	34 ±22	44 ±17	
XV	67 ±18	65 ±15	70 ±14								XIII	55 ±9		
											XIV	55 ±8	60 ±14	67 ±10

II. Alcohol Series.

II	±14	40 ±13	47 ±16	36 ±15	III	70 ±15	70 ±14	42 ±21	42 ±20		V	79 ±10	84 ±8	88 ±12
III	43 ±16	50 ±15	38 ±27		IV	77 ±15	71 ±18	73 ±16	71 ±16		VII	57 ±12	63 ±20	56 ±15
IV	59 ±9	52 ±28	53 ±21		V	74 ±18	75 ±14	74 ±11	75 ±15		VIII	62 ±13	60 ±13	63 ±14
V	59 ±18	75 ±15	60 ±13	65 ±15	VI	70 ±12	74 ±16	81 ±12			IX	63 ±15	65 ±12	63 ±10
VII	70 ±12	68 ±11	67 ±8		VII	73 ±14	76 ±13	71 ±17	73 ±13		X	69 ±15	73 ±13	68 ±11
X	64 ±13	63 ±9	64 ±12		VIII	72 ±19	75 ±18	72 ±16	73 ±14	74 ±16	XI	58 ±9	59 ±11	56 ±12
XI	69 ±9	61 ±15	64 ±13		X	58 ±17	59 ±23	62 ±24			XII	63 ±10	61 ±13	
XII	64 ±10	61 ±15	69 ±11		XI	64 ±12	68 ±19	68 ±19						
XIII	68 ±10	70 ±11	79 ±11		XII	71 ±17	74 ±16	74 ±18						

expressed concerning the general character of the series as given in the first tabulation.

It is desirable to enquire how far the results of this paper resemble those obtained by other observers.

In Exner's experiment one bottle of Hochheimer changed the reaction time from 0·1904″ to 0·1997″, and a second bottle caused a still further slowing to 0·2884″ and later to 0·2969″ or a final change of about 0·1000″[14].

The researches of Dietl and Von Vintschgau are more extended; three experiments were made with the former and four with the latter. They used champagne ($\frac{1}{2}$—1 bottle) or in one case one half litre of "Tiroler Tischwein" which I recall from personal experience as a thoroughly effective combination of alcohol. These authors[15] conclude: If the quantity of wine be not very great the reaction time is shortened for a certain period, and this quickening lasts longer when the wine is drunk slowly in small quantities and also when the amount is more considerable. If a still larger amount (1 bottle) be taken quickly the physiological reaction time is speedily and considerably slowed.

With regard to these inferences several considerations suggest themselves. Unfortunately the probable error is not given and only one "*protocoll*" is printed so that the reader can work it out for himself. The ante-alcoholic averages, too, show so much variation that it is difficult to determine just what change (quantitatively) is produced by the wine. The comparison is commonly made with a large number of old normal observations (326 and 376 respectively) made at various times from which normal averages had been calculated. This method of comparison does not seem to me altogether satisfactory. If we admit its correctness however, it will be seen that nearly all the changes attributed to alcohol are scarcely greater than the differences between "normal" averages on the same day or between these and the general normal, provided we assume the probable errors of the smaller series to be about the same as in the experiments whose details are so fully given as to permit the calculation. In fact only Dietl's marked quickenings in his first experiment (25 June) seem quite trustworthy, and these are no greater than the very unconvincing changes of his third experiment (19 July); and in Von Vintschgau's experiments

[14] I have worked out the probable errors of this experiment, and they are for the above times in order ±0·0033″, ±0·0029″, ±0·0043″, and ±0·0127″, the number of observations being 19, 6, 17, and 5 respectively. Only the last two become incomparable on account of overlapping. The first bottle appears to have improved the trustworthiness of the result as the error is much lessened, but there is a great disturbance of the record under the influence of the second bottle.

[15] Dietl and v. Vintschgau, *op. cit.* p. 382. Their series generally include 17—20 replies. Only one day's work with alcohol is so fully reported as to enable the reader to work out the probable error which varies from ±0·0013″ to ±0·0020″ and is not clearly influenced by the wine.

only the great slowing in the second (22 June) seems above suspicion. In all other cases there are either too few details or the variations among averages independent of an alcohol effect are too great to make any inferences admissible.

Kraepelin used pure alcohol sweetened with a simple fruit syrup. The number of his experiments is large; four persons were tested, the reaction being sometimes a simple one, that is a reply to a sound signal, while in others the more complicated reaction times were taken; in many instances the different reactions were combined. Of course only the simple reaction—i.e. the reply to a simple signal—is suitable for comparison here. Altogether the number of such experiments seems to have been 21 and they were apparently carried out with great care and exactness.

The "average variation" is given in a few cases and several curves are drawn to show the course of the reactions, but these curves are based on the successive groupings of only 3—4 records (which are not given in detail), and single observations appear to be rejected on account of their size as compared with those very near them in the order of the experiment. As I have already suggested this method does not seem to me altogether satisfactory—and the size of the "average variation" leads to the inference that the probable error is really too great to permit all the conclusions which Kraepelin draws. The most constant effect observed was an initial quickening of the reaction time not merely relatively to the preceding series but in many cases the quickening appears to be an absolute one, i.e. as compared with the normal based on a number of "ante-alcoholic" observations for the day, which normal observations I may add show very marked variations. The doses ranged from 7·5 to 60·0 gm. of absolute alcohol. The initial quickening varies in the time both of its appearance and of its duration [3′, 5′, 8′—10′, 10′—20′] and apparently this time increases in some proportion to the slowness of the normal time of the percipient. Where the dose of alcohol was often repeated the relative initial quickening seems to follow nearly or quite every dose. After this initial quickening came "quite constantly" a considerable lengthening of the reaction time. In general this slowing effect was well established in twenty or thirty minutes. For both changes Kraepelin finds an influence of the amount of alcohol the quickening growing smaller and the slowing greater as the dose increases, although the individual effects differ greatly. The slowing influence would also seem to be exerted sooner with larger amounts of alcohol than with small ones.

Kraepelin's results seem to have differed greatly from mine in the amount of change which his doses of alcohol produced. So far as the few printed details enable us to draw any conclusion it would seem that changes of 0·0300″ or even more were quite common as a result of doses which in my experiments had no such effect. Still larger relative changes are quite frequent.

I am convinced concerning my own experiments, as Kraepelin seems to be concerning his, that a much more systematic inquiry is necessary for any final results. If I were to take up the work again I should expect to get more satisfactory conclusions from using small amounts of alcohol, not more than 15—20 c.cm. I should also take more frequent, but regular, records and always in series of at least fifty replies. It would probably also be well to examine the effect of sipping[16] the alcohol—but I feel sure that any effort to vary the dose considerably will lead to no good result unless the number of experiments is much increased beyond anything yet reported. For such work I can heartily recommend the Bowditch Neuramoebimeter. It has proved to be not only convenient and compendious but it also does not easily get out of order, and none of those hindrances from irregular action occur with it which are so often mentioned in reports of work with complicated chronoscopes.

The experiments which I have given with as much detail as seemed reasonable have not been as fruitful as I hoped when I began; they appear to permit the following general conclusions:—

I. The changes in the reaction time after taking varying amounts of pure alcohol are on the whole more considerable than those occurring in equally long experiments without alcohol.

II. There is no obvious and unquestionable relation of the effect, in quality or in quantity, either to the amount of alcohol taken or to the time during which its influence has been exerted.

III. Although the records of A suggest that the reaction time may be quickened shortly after alcohol has been taken, and those of A and of W that the later effect is in the direction of a slowing, such a conclusion is not fairly established by these observations. Nor do the replies of D clearly favour such a view.

IV. Where the normal observations are subject to great variations

[16] Cf. Lauder Brunton's remarks on Cerebral Stimulants in his *Text Book of Pharmacology, Therapeutics and Materia Medica*, 3rd (London) Edition, p. 192.

from day to day (as with D) the alcohol appears to lessen these variations and to bring the observations to a common mean.

V. It is also suggested that alcohol may improve the *quality* of the reaction time, by lessening the errors, at least for a time, but much more numerous series are needed to make this more than a mere suggestion.

NOTES ON MUSCULAR ABNORMALITIES.

By Thomas Dwight, M.D., *Parkman Professor of Anatomy at Harvard University, U.S.A.*

Absence of Flexor Carpi Radialis.—This observation, which I believe is unique, was made in 1876, at the Medical School of Maine, but has never been published. The abnormality occurred on the right side of the body of a muscular young man which I had used for my lectures on myology. The *flexor carpi radialis* was entirely wanting. No trace of anything representing it was to be found at the places of its insertions into the metacarpus. A small pale muscular belly arose from the anterior border of the tendon of the *supinator longus*, about one inch above the styloid process, and (becoming tendinous) passed through a separate sheath near the surface of the annular ligament. The radial artery lay between this extra muscle and the *supinator longus*. I expected to find that this slip took the place of the absent muscle, but it was traced into the *abductor pollicis*. The *palmaris longus* was very strong, its muscular portion extending more than half-way down the fore-arm. Extra muscular slips are not uncommon in this region, as is well known, but I have found no description of one precisely resembling this one. The question arises whether it can be held to represent the *flexor carpi radialis*. The only point in favour of such a view is its position and relation to the artery just above the wrist, but the much more important considerations of its origin and insertion are against it.

Duplicity of the Vastus Externus.—Professor Macalister mentions the fact that this muscle may be double, but gives no details. Testut speaks of it as a very rare condition. Mr Knott writes as follows:—"The vasti have sometimes been found bilaminar, and this is, I think, more frequent than is usually supposed. I have noted five cases of this arrangement in the internal and three in the external muscle, and also met with many others of which I have made no record." In confirmation of this view may be quoted Henle's description of the normal anatomy of the muscle. Mr Williams, in his elaborate

account of the *quadriceps*,[1] shows that the lower anterior edge of the *vastus externus* consists of two tendinous laminæ, between which run nerves and vessels, and which can very easily be accidentally separated in dissection, so that the muscle may be split into two layers. I believe that it is by no means very uncommon to find two layers in this region that do not owe their separate existence to dissection. There is, however, another form of duplicity of this muscle that is due to its arising in two layers. The only case on record that I know of is Gruber's, observed in 1847–8, and published in Virchow's *Archiv*, Bd. lxxxii., 1880. One head arose from the outer side of the great trochanter, from the line to the *linea aspera*, from that line, and from the outer side of the femur half-way down. The other head arose from the upper part of the anterior inter-trochanteric line, from the front of the greater trochanter and from the front of the femur. It was uncovered by the first head in the upper part, but passed under it below and united with it. It was quite distinct from the *crureus*.

I observed two cases in January 1886, which resembled this one in having a double origin, but differed from it in having one head more or less closely connected with the hip-joint.

1*st Case*. Male. White. Left side.—A tendinous band, about one-third of an inch broad and four inches long, arose from the anterior surface of the capsule of the hip-joint a little below the inferior spinous process of the ilium, and joined the deep fascia of the *tensor vaginæ femoris* near the lower end of the muscular fibres. One inch and a half from its origin it gave off a slender tendon, which two and a half inches lower down became muscular, and joined the anterior surface of the *vastus externus* at about the middle of the thigh.

2*nd Case*. Male. White. Right side.—The origin from the *linea aspera* was shorter than usual. The important peculiarity is that a part of the muscle sprang from a tendinous band attached to the anterior inferior spine of the ilium, and apparently representing the inner limb of the Y ligament. The lower part of this was muscular, and joined the anterior edge of the *vastus externus*.

Mr Morris, in his excellent work on the joints, describes

[1] *Jour. Anat. and Phys.*, vol. xiii. p. 204, 1879.

(p. 326) "a tendinous band closely blended with the surface of the capsule stretching between the upper extremity of the tendon of origin of the *vastus externus* and the long tendon of the *rectus*." If this structure is, indeed, of constant occurrence both these cases may be explained as slight variations, but though I have looked for it many times, and have seen the arrangement described by Mr Morris beautifully evident, I cannot agree with him that it is the usual one. It seems to me more common to find a process of fascia passing from the front of the capsule to the deep layer of the fascia lata below the *tensor vaginæ femoris*, and having no connection with the *vastus*.

It occurred to me as possible that in the earlier part of life the muscle might have a higher origin. To determine this, I caused the bodies of some ten or more infants to be examined, but nothing confirmatory was found. I examined myself several fœtuses, mostly of the latter half of pregnancy, but with a negative result, excepting that in one of about four months the *vastus* seemed to encroach on the capsule.

Interclavicularis anticus digastricus.—This very rare abnormality was observed in the winter of 1885–6, on the body of a white woman. The greater part of the clavicular portion of the *pectoralis major* was wanting on both sides. There was only a narrow slip arising at the junction of the outer and middle thirds of the clavicle. This was intimately connected with the deltoid. A vacant space about three inches wide was left between it and the sternal portion. Both the deficiency in the pectoral and the two halves of the abnormal muscle were very symmetrical. The latter sprang from the front of each clavicle by an origin about one inch and a half broad, the outer end of which was covered by the inner border of the clavicular portion of the *pectoralis major*. The outer parts were muscular, and were united by a median tendon about two inches long, which crossed the sterno-clavicular joints and lay in front of the sternum. Some of the superior fibres of the sternal portion of the *pectoralis major* sprang from this tendon.

Professor Gruber[1] has described two cases of this abnormality. It is important to notice that in both of his, as in mine, there

[1] Reichert and Du Bois Reymond's *Archiv*, 1865, and Virchow's *Archiv*, 1878, Bd. lxxii.

was a deficiency in the *pectoralis major*. In the first, the inner part of the clavicular portion was wanting, and the remaining slip was separated by a well-marked interval from the deltoid. In the second case, nearly the inner half of the clavicular portion was wanting, and also the upper part of the sternal portion. In both cases, as in mine, a part of the greater pectoral arose from the abnormal structure. Gruber regards this muscle as representing the union of two sterno-claviculares, one from each side. He reports in the former place an instance of a single *sterno-clavicularis*, in which nearly the whole of the clavicular part of the *pectoralis major* was wanting, and in which the upper fibres of the sternal portion were attached to the origin of the anomalous muscle. I have met with the muscle this year on the left side of a white male subject, which had on both sides a deficiency of the greater part of the clavicular fibres of the pectoral. Professor Macalister wrote in 1871 that Mr Stoker had communicated a case to him, in which the sterno-clavicular muscle coexisted with an absence of the clavicular fibres of the great pectoral. The frequency of this coincidence in the case of both simple and double sterno-clavicular muscles, and the fact that the sternal portion of the pectoral is frequently attached to them, seem to point to some intimate relation between them, and indeed I have presented this group of anomalies on account of the bearing this fact may have on the interpretation of some forms of *sternalis*.

Sternalis.—*1st Case.* Female anencephalous fœtus.—There was a deficiency in each *pectoralis major*. The upper part of the sternal portion was closely united with the clavicular portion. This upper part of the muscle arose on the left from the clavicle, the front of the manubrium, and from the cartilages of the first two ribs. On the right it sprang from the clavicle, the cartilages of the first three ribs, but not at all from the sternum excepting near the sterno-clavicular articulation. The lower portion of the muscle, separated from the upper one by an interspace which is larger on the left, arose on the left from the cartilages of the 3rd, 4th, and 5th ribs, and on the right from those of the 4th and 5th. The *sternalis* was present on both sides. On the right it ran along the inner border of the upper division of the pectoral

from the cartilage of the 4th rib to near the top of the manubrium. On the left the muscle was divided into two parts. The lower ran upwards and slightly inwards from the cartilage of the 4th rib to that of the 3rd, and the upper one from that of the 3rd to that of the 2nd. Its lower part was external to the upper end of the lower one. Both the slips of the left side appeared, judging from a sketch which I made at the time, to partly overlap the pectoral fibres. This case was observed eleven or twelve years ago, before Mr Abraham had called attention to the frequent occurrence of this muscle in anencephalous fœtuses. Since this point was raised I have looked for it in several[1] such cases without once finding it, which is very different from the experience of Mr Abraham and Professor Shepherd. Perhaps sufficient attention has not been paid to the deficiency of a part of the great pectoral on the same side as the *sternalis* in anencephalous fœtuses. Thus, of the six fœtuses in which Mr Abraham found the muscle, four presented a defect in the pectoral. In two of these the defect and the *sternalis* occurred on both sides, in two others on the same side. Professor Shepherd found nine sternales in six anencephalous monsters, and eight times there was a defect of the pectoral on the same side.

I have long been of Professor Bardeleben's opinion that the *sternalis* does not always have the same significance, and that in many cases it stands in some relation to the pectoral. The question now arises whether the occurrence of a defect in the latter muscle does not predispose to an irregular arrangement of other muscular fibres in the neighbourhood, which in some cases may appear as the *sternalis*, and in others as the *sterno-clavicularis*, either single or double, in the latter case being the abnormality described above.

The following cases of *sternalis* were observed in the adult:—

2*nd Case.* Female. Black. Right.—Arises from sheath of rectus opposite 5th or 6th costal cartilage, about 2 inches from median line, runs upward and somewhat inward to end in fascia covering the pectoral at the level of the 2nd cartilage. Breadth about $\frac{3}{4}$ inch.

[1] I can find no record of the number of these observations, but I think I made from four to six.

3rd Case. Female. White. Right.—A triangular muscle; begins by small tendinous bands from sheath of rectus opposite 6th cartilage, 1 inch from median line. The inner border is vertical, slightly concave, the other extends obliquely outwards. Greatest breadth $2\frac{1}{2}$ inches opposite 2nd rib. At this level the outer part becomes fibrous, and ends in fascia covering the outer part of pectoral. It can be traced as a distinct structure for some distance. The inner part runs into the sternal head of sterno-mastoid. *Nerve supply* from intercostals.

4th Case. Male. White. Left.—Arises from sheath of rectus besides sternum opposite 5th and 6th cartilages, and also by (tendinous) fibres that cross the lower portion of the pectoral. Greatest breadth $\frac{3}{4}$ inch. It ends by a narrow tendon in the middle line over the manubrium.

5th Case. Male. South Sea Islander. Double.—This is in some respects a remarkable case. The cartilage of the 5th rib on the right side enlarges to inclose a foramen, and regains its proper size before reaching the sternum. The *right sternalis* began by tendinous fibres from the 7th cartilage just before its junction with the sternum. It was reinforced by others from the 6th cartilage, and by some from the surface of the sternum. It became muscular opposite the 5th cartilage, from which it received additional fibres. Opposite the 4th cartilage it expanded to an inch in breadth, and, growing broader opposite the 3rd, divided into two parts. The outer one spread out over the pectoralis, and was lost in the fascia covering it. The inner division received a minute muscular belly from the 4th cartilage, and soon became tendinous and ran to the middle line, dividing into a slender tendon which joined the sterno-mastoid of the same side, and into several bands which passed under the pectoralis major of the *opposite* side. Some of these were attached to the 2d left cartilage, and two others gave origin to a deep head of the left pectoralis major, which was soon lost in that muscle.

The left *sternalis* arose as a slender tendinous band from the fascia covering the *pectoralis major* at the lower border of the muscle opposite the 5th intercostal space, 1 inch outside of the cartilage. This ran inwards, describing a slight curve till it reached the sternal end of the 6th cartilage, where it turned

suddenly and ran up along the side of the sternum to its insertion into the front of the bone at the level of the 2nd cartilage, where it ended under cover of the right sternalis. *Nerve supply* from a long slender nerve which entered the deep surface of the muscle opposite the 3rd intercostal space, and which was traced through the pectoralis major nearly to the border of the axilla. As the subject was to be used for lectures on topographical anatomy, and as it was undesirable to open the axilla, the nerve was left at this point to be pursued later, which unfortunately was not done. The left nerve was smaller than the right one. They probably came from one of the anterior thoracic nerves.

6th Case. Male. White. Right.—Arose from pectoral fascia over 5th cartilage, about 1 inch from median line, by a narrow flat tendon. The greatest breadth of the muscle was less than $\frac{1}{2}$ inch. It received some fibres from the fascia at its outer edge, and ended in the sterno-mastoid.

7th Case. Female. White. Double. *Right side*—Origin from pectoral fascia over 5th rib, 4th cartilage, and fascia over sternum. Insertion into right sterno-mastoid. Innervation by 3rd intercostal nerve. *Left side*—From pectoral fascia at level of 6th cartilage to left sterno-mastoid. *Innervation* by 2nd and 3rd intercostals.

ERRATA.—In Professor Dwight's paper on "Abnormal Spines," vol. xxi. p. 539, line 21, 1887, for "painful" read "fanciful." It should also be understood that the cervical ribs described on p. 542 belonged to the spine which possessed the abnormal atlas.

(Extracted from the American Naturalist, July, 1887.)

THE RANGE OF VARIATION OF THE HUMAN SHOULDER-BLADE.

BY THOMAS DWIGHT, M.D.[1]

THE late Professor Broca read a paper before La Société d'Anthropologie in 1878[2] in which he described the scapular and the infra-spinous indices, and proposed them as methods of ethnological research. Briefly stated, the scapular index is the proportion of the breadth of the scapula, measured along the base of the spine, to the length, the latter being considered one hundred. The infra-spinous index is the proportion of the breadth to the length of the infra-spinous fossa, the latter being considered one hundred.

The human scapula being the starting-point, Broca understands by "length" the line connecting the highest and lowest points, although in almost all mammals this is not the longest dimension. The line showing the breadth being called AB and that of the length CD, the index is obtained by calculating $\frac{100 \times AB}{CD}$. The length very rarely coincides with the posterior border of the scapula, but runs some distance before it. The line AD representing the infra-spinous length runs from the posterior end of AB to the lower end of CD. The infra-spinous index is represented[3] by the fraction $\frac{100 \times AB}{AD}$. Professor Broca used these indices both in comparative anatomy and in the study of characteristics of race, sex, and age in man.

In all orders of mammals, with one exception, the scapular index is greater than in man. In quadrupeds it is evident that the long diameter of the bone should at least approximately coincide with the line of pressure, and consequently we find the breadth—*i.e*, the line along the base of the spine—the longer. In erect man, with his great range of movement of the anterior extremities, there is need of greater leverage for the movements of the scapula, and less need of resistance to pressure, so we find a long and narrow scapula. This condition is approached in the anthropoid apes, and even surpassed in the bats, who have a

[1] Parkman Professor of Anatomy at Harvard University.

[2] Bulletins de la Société d'Anthropologie de Paris, série 3, tome i.

[3] By a most unfortunate oversight the AB and AD are transposed in Broca's paper.

lower scapular index than man. Broca's observations on the characteristics of sex and race of human scapulæ are by no means satisfactory, owing to the small number of observations. The indices of adult Frenchmen were obtained from twenty-three individuals, of which nine were female; and those of African negroes from twenty-five individuals, of which five were female. Apparently one scapula was measured in each case.

The next year Professor Flower and Dr. Garson measured two hundred scapulæ of Europeans. They admit that it is quite possible that some of the bones belonged to members of other races, and they paid no attention to sex, which for the most part was unknown. They counted every scapula measured as one, whether or not its fellow came under observation. They measured also a few scapulæ of other races, but the series were for the most part very small. The largest were twenty-one Andaman scapulæ and twelve Australian.

M. Marius Livon also studied this subject at about the same time, and gave his results in his "Thèse pour le Doctorat," which appeared in 1879. I have never seen his essay, and know it only by references to it by Sir William Turner[1] and by M. Manouvrier.[2] He measured the bones of seventy-three Frenchmen and fifty-one Frenchwomen.

Negroes, Andamanese, and Australians have higher indices, which means broader scapulæ, and consequently of a lower type. Broca points out, however, in his remarks about negroes, that this is true only of the mean, and that individuals of both classes are found to vary very much from it. Indeed, the anthropoid apes may have at least a scapular index within the range of the variations of man. The greater obliquity of the spine in apes makes their infra-spinous angle more characteristic.

Broca claimed that, in spite of the great individual variation, this method is of value when applied to groups.

I began a series of measurements of human and of anthropoid shoulder-blades several years ago and put it aside for other work. I have recently resumed it. My original purpose had been simply to collect additional statistics, but as I went on I was much struck by the diversity of forms which the scapula presents, as well as by the variation of the indices. I became convinced both that

[1] "Challenger" Reports, vol. xvi.

[2] Revue d'Anthropologie, 2me série, tome iii., 1880.

the indices do not necessarily indicate the shape of the bone, and that they are worthless to determine the race of any single bone.

The one hundred and thirteen bones which I have called Caucasian are, like those used by Flower and Garson, rather a heterogeneous collection. They for the most part belong to the Harvard Medical School and to the Boston Society of Natural History. More than a few of them came from France. While they, no doubt, are in the main Caucasian, it is probable that there are some negro bones among them. Indeed, I know that two of the scapulæ came from the body of a negro. These are remarkable as presenting very low (instead of high) indices.

Through the kindness of Professor Putnam, who most generously put all the stores of the Peabody Museum of Archæology at my disposition, I had hoped to be able to present a large collection of figures from the bones of the mound-builders, and perhaps to make observations on many individuals of a race which was less mixed than most of those whose bones are easily obtained to-day. I was, however, disappointed, and from a cause that is easy to foresee, namely, the great fragility of the scapula. I could have had long bones in abundance, but the shoulder-blades were for the most part either in fragments, or so injured that the necessary measurements could not be made. I have the records of six scapulæ from California which are probably Indian, and of eighteen from the Kentucky mounds. The mean of the Californians is 67.25 for the scapular index and 91.05 for the infra-spinous. The mound-builders have a mean scapular index of 69.29 and an infra-spinous one of 93.75.

The following table shows the indices of Caucasian bones obtained by my predecessors and myself. Livon is the only one who has a tolerably large series showing the difference between the sexes. Broca, with a much smaller series, had different results:

TABLE I.

	Broca.	Flower and Garson.	Livon.		Dwight.
			Male.	Female.	
Scapular index	65.91	65.2	63.0	67.4	63.5
Infra-spinous index	87.79	89.4	85.4	88.8	85.8

To understand the significance of figures something more is needed than an average, which gives no hint of the range of variation, and accordingly I have arranged the figures of the in-

dices of the Caucasians and mound-builders in groups. The first row of figures shows the index, and opposite each is placed the number of scapulæ having that figure (or that figure and a fraction) for an index. The six Californians showed no extreme figures.[1]

TABLE II.

Scapular Index.			Infra-spinous Index.		
Index.	Caucasians.	Mound-builders.	Index.	Caucasians.	Mound-builders.
55	2	...	72	2	...
56	3	...	73	...	...
57	5	...	74	...	...
58	7	...	75	1	...
59	5	...	76	2	...
60	9	...	77	4	...
61	8	...	78	4	...
62	12	...	79	8	...
63	13	1	80	7	...
64	6	...	81	4	...
65	11	2	82	6	...
66	10	3	83	5	...
67	8	2	84	4	...
68	1	3	85	10	...
69	4	1	86	7	...
70	3	1	87	10	...
71	3	1	88	5	3
72	1	1	89	3	3
73	1	...	90	8	3
74	...	...	91	1	...
75	...	...	92	6	2
76	1	1	93	2	1
77	...	1	94	5	...
78	...	...	95	2	1
79	...	...	96	3	1
80	...	...	97	1	1
81	...	...	98	1	...
82	...	1	99	...	...
			100	1	...
			101	...	...
			102	1	...
			103	...	...
			104	...	...
			105	...	1
			106	...	1
			107	...	1
Total.....	113	18	Total.....	113	18
Mean.....	63.50	69.29	Mean.....	85.83	93.75

[1] There can be no question that in measurements the "personal equation" has its effect. No doubt the discrepancies would have been less if all the measurements had been made by one man. The very greatest care is needed to avoid mistakes. I was much mortified at the conclusion of this work to find inaccuracies in the measurements of two bones, the results of which it was practically impossible to rectify. Happily the errors are of little consequence.

The scapulæ from Kentucky have a decidedly higher index than the Caucasians, and this is evidently not due to individual peculiarities, for though three specimens with remarkably high indices have their effect on the average, yet the scapular index is only once, and the infra-spinous never, as low as the Caucasian mean.

Broca gives some of his extreme figures, which are very interesting. The bone with the lowest indices belonged to a Turk from Smyrna; the next lowest scapular index was furnished by a Frenchwoman, and the next lowest infra-spinous by an Arab. The highest indices belonged to an African negro, by name Tom Blaise, and the next to a black from Hindoostan.

Flower and Garson, unfortunately, make no mention of their extreme cases. For more convenient comparison I put my extremes below Broca's in the following tables:

TABLE III.

MINIMA.

	Scap. Index.	Infra-spin. Index.
Broca. Turk from Smyrna	57.47	75.19
Broca. Frenchwoman	60.27	.....
Broca. Arab	.....	78.57
Dwight. Caucasian (Fig. 4)	55.1	72.8
Dwight. Negro (Fig. 10)	58.8	72.3

MAXIMA.

	Scap. Index.	Infra-spin. Index.
Broca. Tom Blaise	76.64	111.95
Broca. Black from Hindoostan	76.61	104.39
Dwight. Kentucky Mound-builder (Fig. 5)	82.2	107.3
Dwight. Kentucky Mound-builder	77.1	106.9
Dwight. Highest scapular index among Caucasians	76.3	98.2
Dwight. Highest infra-spinous index among Caucasians	71.8	102.0

Broca thought it likely (*assez probable*) that his limits would be but rarely passed. Table II. shows, however, that I have measured no less than twenty-two bones with a scapular index below sixty, and that five of them were below fifty-seven. The two given in Table III. are, however, the only ones in which the infra-spinous index falls below seventy-five. Turning to the *maxima*, I have no equal to Tom Blaise in both indices, which is due to the great obliquity of the spine of his scapula, an ape-like feature which the mound-builders cannot rival. One of

them has a scapular index which is decidedly higher, and they both exceed the black from Hindoostan in both respects.

Sir William Turner states in his "Challenger" report that, excluding the scapulæ of one Hottentot, the mean scapular index of several races ranged from 60.3 in the Tasmanians to 70.2 in the Andaman Islanders. This includes the results of some other observers. It would appear, however, that for some reason he has omitted also his four Ohauan (Pacific Islands) scapulæ, which have the extraordinary *means* of 78.8 for the scapular index and 117 for the infra-spinous. He found the variations in the scapular index of African blacks, male and female, to range from 57 to 81, and the infra-spinous index from 80 to 117.

Apart from the range of individual variation in the indices, this method is open to at least two criticisms: first, that there are various forms of scapulæ, which may not be without their ethnological significance, to which these indices give no clue; and, secondly, that the length of the scapula—which is of primary importance in determining the more important index, the scapular—depends in part on the development of the superior angle of the bone. In support of the first criticism I would call attention to two scapulæ (Figs. 6 and 7) whose indices are almost identical, yet which in shape differ enough to be bones of different species. I shall return to these points in the course of the discussion of the variation of different parts of the bone.

Length.—In the one hundred and thirteen Caucasians, all adults, the mean length is 16.22 cm., the extremes being 13.2 and 20.1. There are six under 14 cm. and ten of 18 or more. The mean of the six Californian Indians is 13.62, and of the eighteen from the Kentucky mounds 14.07. The range of variation in these two series is very small. The shortest bone is 12.4, and the longest 15.8, both from Kentucky. These old bones, both in size and shape, constitute a well-marked series.

Professor Mivart, in his well-known paper on the "Appendicular Skeleton of the Primates,"[1] takes several parts of the scapula for comparison. We shall consider the variation that some of these present in man alone.

The inferior angle (Fig. 1),[2] which Professor Mivart puts at

[1] Philosophical Transactions, London, vol. clvii., part 2, 1867.

[2] In Figures 1, 2, and 3 the partial outlines are drawn as though taken from the bone of the right side in every case. This is for convenience.

35° or 40° in man, presents great differences, as is shown by the appended wood-cuts. I give no measurements, as the difficulty of making accurate ones is quite out of proportion to their value. The difference is in part, but not wholly, due to the development of the surface at the lower part of the axillary margin for the teres major.[1] This muscle arises from the dorsal surface of the bone, but there is almost always a slight projection at this point from the anterior border, and occasionally it is developed into a projection of considerable size. Broca states that this is more common in negroes. I have no opinion to offer on this point, but I believe that this process does not stand in direct ratio to the size of the muscle. It is well marked on a very delicate scapula (Fig. 5), and on the other hand there is a large surface to this muscle with hardly any projection on a remarkably sharp bone (Fig. 4). An analogous case is the third trochanter, the occurrence of which, in my opinion, is quite uninfluenced by muscular development. The surface for the *teres major* projects out very strongly in the lower Simiidæ, Cebas, and Chrisothrix, according to Mivart. Its occurrence in man is probably (as in the case of the third trochanter) the appearance of a peculiarity of lower forms. The lower angle, however, varies considerably apart from the influence of this process.

FIG. 1.

The vertebral border (Fig. 2).—The most common form of scapula presents a line slightly curved at the lower part, and then straight as far as the root of the spine, from which point it inclines slightly forward till it ends at the upper angle. The forward inclination of the upper part, though varying in degree, is, so far as I know, constant, but the rest of the line varies much. Sometimes it is almost straight, sometimes the whole border of the bone is convex, sometimes the border below the spine is con-

[1] By another oversight Broca repeatedly calls this muscle "*le petit rond.*"

cave. The different types are seen in the annexed diagram, but, better still, in some of the figures of the entire bone.

The superior border (Fig. 3) is also of uncertain shape. The superior angle cannot be considered apart from the posterior border. The diagrams show its variations so well as to make a description unnecessary. One of these is remarkable as showing the point truncated. None of these, however, show it rounded off in a way that equals the Boschiman scapula figured by Mivart. The variations of this angle are important, because they show that the length of the scapula, as used according to Broca's plan, is liable to vary according to the development of this angle. The

FIG. 2.

FIG. 3

bone with the truncated angle has of course a scapular index decidedly different from the one it would have if the posterior and superior borders had been prolonged till they met in a sharp point. The differences in depth of the supra-scapular notch, which ranges from the merest deviation of the course of the superior border to a deep notch and then to a foramen, are well known. I have one specimen in which there is a foramen below a deep notch that is almost bridged over. It is the only case of the kind I have ever seen. The only allusion to its occurrence that I know of is in Humphry's "Human Skeleton."

Variations in the concavity of the body of the bone should also be mentioned. If a scapula be laid on a table with the ven-

tral surface downward, it will usually rest on three points,—the coracoid, and the superior and inferior angles. The vertebral border, as a rule, forms an arch, the highest point of which is sometimes about 2 cm. from the table. In other cases this border barely leaves the table, and sometimes one or both of the angles mentioned are bent dorsally, so as no longer to be points of support.

On the dorsum the course of the spine and acromion deserve attention. By comparing the scapular and infra-spinous indices we get some idea of its obliquity. A high infra-spinous index with a moderate scapular index shows, of course, a greater obliquity, which is an ape-like peculiarity. When both are very high it shows simply a short, broad scapula. I called attention to the fact that one of the Kentucky mound-builders had a higher scapular index but a lower infra-spinous one than Broca's negro, Tom Blaise, which means that the former had the relatively broader scapula, but the latter the more oblique spine. A study of the one hundred and thirteen Caucasian shoulder-blades shows that the two indices present no great discrepancies. The average scapular index being 63.50 and the infra-spinous 85.83, I find but three with a scapular index of between 64 and 65 which have an infra-spinous index below 85, and of these the lowest was 82.5. I find none at all with a scapular index above 65 and an infra-spinous below 85. Conversely, only seven having an infra-spinous index above 86 have a scapular index below 63. The lowest of these is 61.

Professor Mivart states that when the bone is so held that the long axis of the glenoid cavity is vertical, and that cavity is opposite the eye of the observer, the acromion is almost always higher than the coracoid in man, troglodytes, and hylobates. I do not remember any exception to this rule in man, but I find a good deal of variation in the direction of the line formed by the projecting edge of the acromion when the bone is thus held. It is not easy to determine what angle it forms with the horizon, and I shall give the mean very vaguely, as probably in the neighborhood of 45°, but I have seen it reach at least 65° on one hand and fall to 30° or less on the other. These two extremes are shown in Figs. 12 and 13. I have been unable to find that either of these degrees of inclination is associated with any particular shape of the bone.

The study of the range of variation in these separate parts leads one to the conclusion that very remarkable scapulæ could be constructed by a judicious selection and union of the most striking individual parts; but it would, I think, be difficult to make more remarkable ones than actually occur. I shall now call attention to some of the most curious specimens. Figs. 4 and 5 show respectively the lowest and the highest indices. The breadth is the same in each, but the length of the latter is little more than two-thirds of that of the former. The process for the teres major, though small, is clearly shown on the scapula of the mound-builder, but the other bone has a larger surface for the origin of the muscle, though there is but a slight projection at the anterior edge, which is confirmatory of the views expressed above. Figs. 6 and 7 have already been alluded to as widely different forms, having almost identical indices. Each is a peculiar bone, but the sharp one is the more uncommon. Figs. 8 and 9 are also in strong contrast to each other. The posterior borders have been figured, but the striking effect is shown in the figures of the whole bones. Each is very peculiar and in a different way, though neither has a remarkable index. Fig. 10 represents a wonderfully long bone,[1] being 20 cm. It is a remarkable specimen, and is among those having the lowest indices. The scapular index is 56.5, and the infra-spinous 76.3. The original of Fig. 11 (scapula index 58.8 and infra-spinous 72.3) is figured because it belonged to a negro and yet had indices far below the mean of Caucasian bones. A comparison of these bones shows assuredly remarkable diversities of form. It is easy to find bones of different species of animals that differ less from one another than do these human ones. I am unable to say what remarkable forms might be seen in a large collection of the shoulder-blades of the Felidæ, but I know that a small collection shows no such range. Even the scapula of the bear differs no more from that of the tiger than some of these from one another. A few notes on the anthropoid apes will be found in the appendix.

What influence the height, the muscular development, the health, and the occupation of the individual may have on the shape of the scapula, and indeed on the skeleton in general,

[1] The mate to this bone is the longest I have measured, exceeding it by more than 1 mm., but it has, unfortunately, been injured.

PLATE XX.

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6

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PLATE XXI.

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11

12

13

cannot as yet be much more than guessed at. The question suggests a field for inquiry, which has as yet hardly been opened, but which in the course of years may bear a rich crop. Something has been done in this direction by Mr. W. Arbuthnot Lane, of London.[1]

Livon found that in women both indices were higher than in men, while, as above stated, Broca found the reverse, but in very small series. I do not feel convinced that Livon's are large enough to put the fact beyond question, and certainly it would be rash to draw any conclusion as regards a single individual. Probably the actual size of the bone, and more particularly that of the glenoid cavity, are the best indications of the sex that the bone offers, and these are of but little value.

It must be remembered that the great range of individual variation does not necessarily destroy the value of Broca's indices, but it shows that the method must be applied to large series of bones from well-marked races. It is gratifying to find this view supported by no less an authority than Sir William Turner, who writes, "For I gather from my own measurements and those of other observers, that the range of variation in the relative length and breadth of the scapula is very considerable in the same race, so that it needs a large number of bones to enable one to obtain an accurate idea of the mean of any race."[2]

APPENDIX.

THE INDICES OF ANTHROPOID APES.

Both Broca and Flower and Garson published the indices of anthropoid apes in their papers, which have been referred to so often. As the number of observations is necessarily small, I am glad to be able to offer a few additional ones. Broca gives the indices of ten gorilla skeletons, of five chimpanzees, of one orang, and of seven gibbons. Flower and Garson give the indices of sixteen bones of the gorilla, of twenty-one of the chimpanzee, of seventeen of the orang, and of eight of the gibbon.

[1] The Pathology of Changes produced by Pressure in the Bony Skeleton of the Trunk, Guy's Hospital Reports, vol. xliii., 1886.

[2] It is proper to mention that this paper had been written and sent to the NATURALIST before I had seen Professor Turner's remarks on the scapula in the "Challenger" Reports. Through the courtesy of the editors I have had an opportunity to modify my paper here and there by quoting from his work.

To these I can add the indices of thirteen shoulder-blades of the gorilla, eight of the chimpanzee, and two of the orang.[1]

	Broca.		Flower and Garson.		Dwight.	
	Scap. Ind.	Inf-spin. Ind.	Scap. Ind.	Inf.-spin. Ind.	Scap. Ind.	Inf.-spin. Ind.
Gorilla.....	70.38	126.05	72.2	132.5	70.1	131.7
Chimpanzee...	68.52	130.23	69.9	133.8	68.4	129.1
Orang.....	69.27	97.46	77.6	103.8	73.6	89.7
Gibbon	96.97	198.56	96.5	201.2		

Below are the highest and lowest indices that I found in gorilla and chimpanzee:

		Scapular Index.	Infra-spinous Index.
Gorilla	Highest.....	76.6	153.3
	Lowest.....	66.1	116.0
Chimpanzee...	Highest.....	72.8	136.0
	Lowest.....	66.1	117.5

These figures show that the highest scapular index of the gorilla and chimpanzee is sometimes, though very rarely, surpassed in human bones. I say nothing of the orang, having measured the bones of only one adult skeleton. The case is different, however, with the infra-spinous index, as the lowest of the gorilla and chimpanzee exceeds the highest human one.

Description of Plates.

The views of the dorsum of the scapula are all on the same scale. Figures 12 and 13 are not on precisely the same scale as the others. Figures 6 and 13 are from the same bone. All but 5 and 11 are presumably Caucasian.

		Scapular Index.	Infra-spinous Index.
Fig. 4.	.....	55.1	72.8
" 5.	From Kentucky mounds.....	82.2	107.3
" 6.	.....	67.8	91.4
" 7.	.....	67.8	90.6
" 8.	Two opposite forms.....	69.4	97.2
" 9.		61.5	91.4
" 10.	A very long bone.....	56.5	76.3
" 11.	Negro.....	58.8	72.3
" 12.	Show extreme degrees of inclination of the acromion.		
" 13.			

[1] In spite of a foot-note on p. 75 of Broca's paper, I confess to some doubt as to whether his figures of ape measurements refer to the number of skeletons or to the number of scapulæ. I have followed Flower and Garson, who reproduce his table, in stating it as I have in the text. My own figures, like theirs, refer to individual bones.

July 23, 1887.

THE PROCESS OF REPAIR

AFTER

RESECTION OF THE INTESTINE

AND

INTESTINAL SUTURE.

BY

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OF BOSTON, MASS.

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THE PROCESS OF REPAIR AFTER RESECTION OF THE INTESTINE AND INTESTINAL SUTURE.

By J. COLLINS WARREN, M.D.,
OF BOSTON, MASS.

The following experiments were performed chiefly for the purpose of studying the process of repair as exemplified by the peculiar conditions which surround a wound of the intestinal walls, but also with the hope of contributing something of interest, and, perhaps, of value, to the surgeons and investigators who are now occupied with this interesting field of surgery.

The operations upon animals were performed in the Physiological Laboratory of the Harvard Medical School. Strict asepsis was not attempted in all cases, partly because the tolerance of the peritoneum of dogs is so great that precautions are not necessary to secure prompt union of the bowel and abdominal walls, and partly because it was desired to observe the effects of localized inflammation upon the function of the sutured intestine.

As a rule, the abdomen was shaved and scrubbed with a one per cent. solution of carbolic acid, or a one to five thousand solution of bichloride. The animal was covered with towels wet in the bichloride solution, and the portion of abdominal wall left uncovered was sprinkled with powdered iodoform. Sponges were kept in a hot bichloride solution, and instruments in the carbolic solution.

Females were usually selected for these experiments, as it would have been well-nigh impossible to keep an aseptic dressing upon this region of a male.

The omentum was either drawn up, or an incision was made

directly through it. The bowel to be resected having been drawn out through the wound, was held by the hands of an assistant, and protected with hot cloths and sponges. The segment selected for removal was about two to three inches in length usually, and included the area supplied by one mesenteric branch. A ligature was placed around this vessel at its origin, and the intestine and a V-shaped portion of omentum were severed by the scissors. The anastomosing branches were then secured by ligature. These branches can be also conveniently tied before resecting the bowel.

The material used for the suture was the finest sewing silk, armed with a needle fitting closely upon it.[1] The needle is better short, as an unduly long needle is apt to bend or break. As it is not the purpose of these investigations to test the value of any particular form of suture, it will not be necessary to refer to the different varieties that have been invented from time to time.[2] The "interrupted Lembert" was used in nearly all the experiments. On two occasions a suture devised by Dr. H. W. Cushing, of Boston, which he describes as the "right-angled continuous suture," was employed.

It was not attempted in all cases to penetrate the submucous fibrous layer, as recommended by Halstead, and in but one instance was there a failure to hold the wound, by which is meant that one only of all the stitches taken failed to hold. There did not appear to be any difficulty in avoiding perforation of the mucous membrane; no signs of such an occurrence were observed in any of the specimens.

The first stitch was taken through that portion of the bowel which had no peritoneal covering, namely, that portion lying between the reflected layers of the mesentery. The sutures were continued around the bowel at intervals of about three mm. The mesentery was then sewed with the ordinary interrupted suture.

Suture of the intestine, as has probably been observed by every experimenter, is much more difficult in dogs than in the

[1] "Parabola gold-burnished Sharp's, No. 10."

[2] Boston Med. and Surg. Journal, January 6, 1887. Dr. Otis K. Newell.

human subject, owing to the tendency to extreme eversion of the mucous membrane, due to the powerful retraction and contraction of the longitudinal and circular muscular layers. This is, at times, sufficient to close the lumen of the bowel so that the contents escape with some difficulty. After a little experience, fifteen minutes were found amply sufficient to perform the suture.

Occasionally it was found necessary to reinforce the sutured wounds at one or two points, but as this involves the curling in of a deeper rim of inverted wall, it is as well to make the first row of sutures as accurate as possible.

The suture complete, the bowel was carefully washed and returned as nearly as possible to its natural position in the abdominal cavity.

No suture of the omentum was made in case it had been cut through. The abdominal walls, including all the layers, were then brought together with stout silk sutures, about eight in number, and the edges of the wound were more accurately adapted here and there with a few superficial sutures. The wound was then dusted freely with iodoform, after a thorough washing, and was either allowed to remain uncovered or was dressed with borated cotton, held firmly in place by a double spica bandage, which was firmly sewed together. This dressing was not changed for a week, but iodoform was dusted on daily around its edges.

In all cases the wounds united by first intention; there was, however, a tendency to suppuration about those sutures which were uncovered by a dressing. No difficulty was found in getting good antiseptic results when all precautions were carefully followed out.

The treatment consisted in starvation for three days, followed by an almost exclusively milk diet.

The following cases have been selected to show the process of repair, under varying conditions, during six months following the operation. In no case did the animal die of the operation.

THREE DAYS.—Bull terrier bitch, medium sized; no antiseptic precautions; about one inch of small intestine resected. The continuous Lembert suture was used, one needle and thread only being employed for intestine and mesentery. A little iodoform was dusted on before and after closing the incision in the abdominal wall. The animal was fed on milk, and did well, bowels acting normally; in three days the animal was destroyed. The wound of the abdominal walls was clean and without discharge, having been kept in good condition by the animal itself; on pressure, a drop or two of slightly reddish serum could be pressed from the openings of the sutures. The walls were firmly united; the bladder, which was distended at the time of the operation, was adherent to the inner surface of the wound.

The sutured point was concealed by a mass of indurated and matted omentum, which laid over it and glued together several adjacent coils of intestine. No sign of the sutured mesentery could be seen. The intestines, though coiled and twisted together, admitted water to flow freely through the lumen.

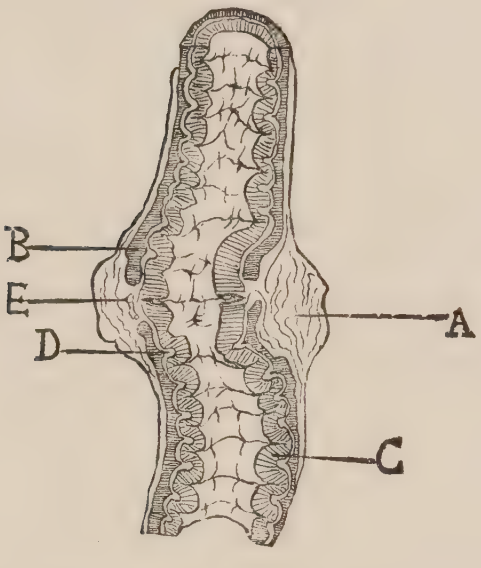
EIGHT DAYS.—Medium-sized mongrel bitch. Fair antiseptic precautions; incision in median line; omentum drawn up, and two inches of intestine resected. Ends of intestine united by Dr. Cushing's continued suture. It is interesting to note that, after this suture is completed, no sign of the thread can be seen. A continuous suture of mesentery was also made. An antiseptic dressing was applied after closure of abdominal wound (iodoform, boracic cotton, and bandage).

At the autopsy the omentum was found adherent to and concealing the line of suture; slight matting together of an intestinal loop by a mesenteric adhesion. The lumen was pervious, and union of the sutured intestine was firm and complete.

EIGHT DAYS.—Medium-sized mongrel bitch. Resection of about four inches of intestine, with corresponding mesentery. Ends of intestine united by interrupted Lembert stitches, and mesentery united by continued suture. The animal had no bad symptoms.

At the autopsy it was impossible to find the exact seat of suture, owing to an extensive matting together of the intestines, which were coiled together in a large lump around the resected spot. It was only after much tearing apart and cutting of the adhesions that the point of suture could be found. The specimen (Fig. 1) from this animal shows a thickening of the peritoneal covering over the line of excision in the form of a callus.

FIG. 1.



LONGITUDINAL SECTION OF INTESTINE EIGHT DAYS AFTER SUTURE, SHOWING AN EXTERNAL CALLUS. THE MUSCULAR COAT HAS RETRACTED SLIGHTLY, AND NO INTERNAL RIDGE IS SEEN.

A. Thickened peritoneum.
B. Muscular coat.
E. Suture.
D. Fibrous coat.
C. Mucous coat.

FOUR DAYS.—Longitudinal wounds were made in the intestine of a dog at short distance from each other, two of these being linear incisions, and the third made by cutting out with curved scissors a portion of the intestinal wall. One was united with the interrupted Lembert suture, the other by continued sutures. On stitching the last wound the calibre of the intestine was greatly reduced in size, so that an instrument not larger than a No. 12 English catheter could have been passed through it.

Considerable matting together of the intestines was found at the autopsy, but the lumen at the narrow point seemed to have been restored. On laying open the intestine threads of the continuous suture were found hanging from the wound into the intestinal canal. There was a hole in the intestinal wound from which a portion of the intestine had been removed extending through the walls of the bowel. There was no protrusion of the coat of the intestine inward; on the contrary, a distinct furrow was found in the mucous membrane marking the line of the wound in each case.

Nine Days.—A large bitch; antiseptic precautions. Incision through the omentum. About two inches of intestine resected, together with a V-shaped portion of mesentery. Mesentery first sewed up with continued suture, and the intestinal ends united by an interrupted Lembert suture. The animal suffered considerably, necessitating two subcutaneous injections of morphia, after which there were no bad symptoms; the bowels acted well before it was destroyed.

No signs of adhesive peritonitis; the line of the suture in the mesentery was distinctly seen, and the intestine at the point of suture was easily separated from the overlying omentum. There was no narrowing of the calibre of the intestine. At the point of suture a faint wrinkle merely of the mucous membrane marked the line of the incision.

The specimen shows the results obtained by a carefully adjusted suture, not including too much of the intestinal wall, and the careful asepsis preserved.

Eighteen Days.—Irish setter; antiseptic precautions. An incision was made in the median line, the omentum drawn up, and a piece of intestinal wall about one inch long and three-quarters of an inch wide was excised with curved scissors. The edges of the wound were united longitudinally with the continuous Cushing suture; owing to the retraction of the circular fibres the calibre of the sutured gut was reduced to the size of a pen-handle when returned into the abdominal cavity. Two inches lower a smaller piece was removed, and the edges brought together transversely by the same suture. Antiseptic dressing, which was changed on the eighth day, when the stitches of the abdominal wound were removed, it having healed firmly.

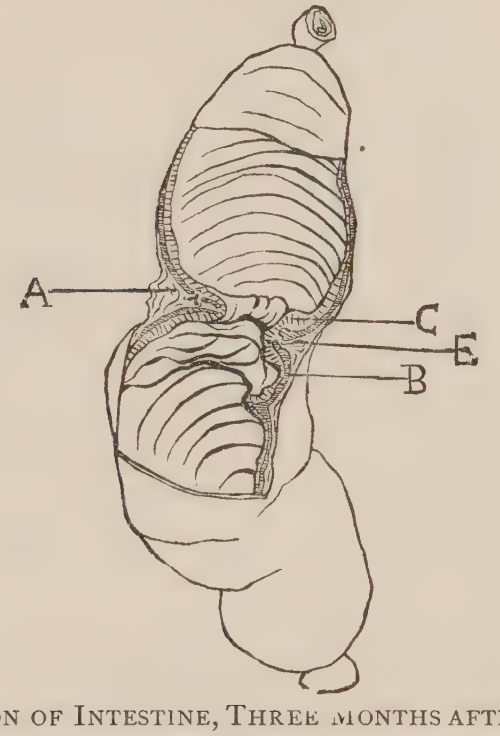
The animal was destroyed on the eighteenth day, being in good condition, passing feces, and feeding well. An adhesion of the omentum was found at one point of the intestinal cicatrix. Some adhesions were found on the mesentery, which folded that part of the intestine, including the two wounds, into a long, oval loop. The narrowing of the first wound still remained nearly the same as at the time of suture, and there was a distention of the bowel immediately above by accumulated food. The other wound had united so neatly that the size of the intestine was perfectly preserved, and it was with some difficulty discovered.

Three Months.—Large-sized, male pointer; antiseptic precautions taken, but no attempt at antiseptic dressing to the wound. About

three and a half inches of intestine were resected at a point about twelve inches from the cæcum, and a corresponding piece of mesentery. Interrupted suture to the intestine, and continued suture to the mesentery.

The wound united well, slight suppuration occurring from the tracts formed by some of the sutures, which rapidly healed on their removal. For some weeks after the operation the animal appeared to suffer pain, and barked almost continuously, at times vomiting food. These symptoms, however, gradually disappeared, digestion became good, and the bowels regularly moved by normal fecal evacuations. It was noteworthy that, though during the last two months of life its appetite was voracious, it did not gain flesh.

FIG. 2.



LONGITUDINAL SECTION OF INTESTINE, THREE MONTHS AFTER SUTURE. A RIDGE OR DIAPHRAGM NARROWS THE CALIBRE OF THE INTESTINE AT THE POINT OF SUTURE.

A. Peritoneal coat.
B. Muscular coat.
C. Mucous coat.
E. Point of suture.

At the post-mortem examination the omentum was found slightly attached at the lower portions of the abdominal cavity; one of the adhesions was attached to the intestinal cicatrix, which was further obscured by a matting of the intestines, not only of immediately adjoining portions of the tube, but also of a loop some twelve inches

nearer the stomach. This mass of matted intestines was in close proximity to the cæcal valve, which, however, was not involved. The mesenteric cicatrix was concealed by the matting of the parts.

On opening the intestine at the point of cicatrization, a high ridge was found encircling the wall, at some points higher than at others. The appearances suggest an unintentional increased inversion of the intestine as the suture proceeded. The diaphragm found at this point, and the extensive adhesions of the intestines no doubt accounted for the symptoms of obstruction which were present during the weeks following the operation.

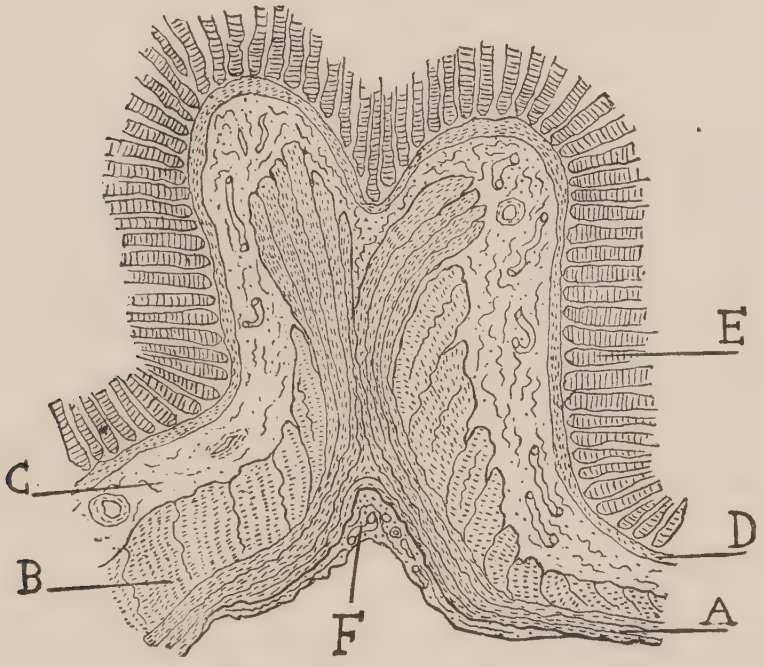
A portion removed for microscopical study showed a proliferation of the peritoneal layer, and also an activity of the cells in the muscular layer. The mucous membrane had closed over the edges (Fig. 2).

Six Months.—Good-sized spaniel (male). The intestine was resected at a point about one foot from the pylorus; no dressing applied. Eight days later the wound, which had firmly united, was reopened to establish the fact of a localized matting of the intestines. On introducing the finger into the abdominal cavity, a hard, irregular, nodulated mass was felt, which was with difficulty lifted from its bed out of the abdominal cavity, it being necessary first to divide numerous omental adhesions. The mass was found to consist of a coil of intestine so adherent that the point of ligature could not be seen. The mass was returned, and the wound closed again. There appeared to be no intestinal obstruction, it being possible to squeeze gas from the intestine above to that below the point of ligature. There was also no appreciable distention at any point. Six months later the animal, which was in perfectly good condition, was destroyed. The abdomen was opened by a circular incision, which included the cicatrix. On lifting this up the intestines were found uncovered by omentum, which had apparently disappeared. It was found, however, that the entire intestinal canal, from the point of suture to the cæcal valve, had escaped through the opening in the omentum which was made at the time of the operation. The intestine could easily be returned through this opening, and it was then found that the adhesions noted as existing one week after the resection had almost entirely disappeared. The omentum, as in all cases, was adherent to the linear cicatrix in the bowel, and a slight narrowing of one portion of the mesentery was observed, due to a cicatricial contraction. The suture in the mesentery was distinctly seen in its whole length. On distending the bowel

with alcohol, and opening it after hardening, a considerable narrowing of the calibre was found at the point of suture, due to so great inversion of the intestinal walls. Owing to narrowing of the mesentery at one point, a kink was formed, when the intestine was distended, and produced a marked spur interiorly, narrowing the calibre at that point.

A microscopical examination of a portion of the cicatrix, removed for the purpose, showed an intimate blending of the bands of longitudinal fibres of the muscular coat, which suggested strongly a union of this coat by a reproduction of muscular tissue (Fig. 3).[1]

FIG 3.



SECTION OF CICATRIX AT SIX MONTHS (SEE FIG. 2, E). THE VARIOUS COATS HAVE UNITED, EXCEPT THE CIRCULAR FIBRES, WHICH HAVE RETRACTED SOMEWHAT.

A. Longitudinal muscular fibres.
B. Circular muscular fibres.
C. Fibrous layer.
D. Submucous muscular layer.
E. Mucous membrane, shown in part only.
F. Thickened peritoneal layer.

Case of strangulated hernia operated upon by Dr. M. H. Richardson. The hernia was first operated upon five years before by injection for a radical cure. The relief had been complete until the day before the operation by Dr. M. H. Richardson. The sac was opened and

[1] These experiments were performed in the Physiological Laboratory of the Harvard Medical School, with the assistance of Dr. G. H. Monks.

the loop of intestine reduced. The symptoms not being relieved, the abdominal cavity was opened four days later and a reduction *en bloc* found with sloughing intestine. The gangrenous portion was excised with a corresponding portion of the mesentery. The much greater facility with which the suture (the Lembert) could be applied than in the dog was very noticeable, owing to the absence of eversion of the mucous membrane. The patient died twenty hours after the operation. The patient was a female, aged sixty-five. The hernia was inguinal.

At the autopsy it was found that the obstruction which existed during life had not yet been entirely relieved, the upper portion of the intestine remaining distended. Matting of the intestine had also occurred in this case.

Dr. W. Wotkyns Seymour's case of pistol-shot wound of the abdomen.[1] The patient, a boy aged sixteen, was operated upon, about twelve hours after the injury was received, in a basement tenement, but with all the antiseptic precautions. Blood and feces were found in the abdominal cavity. Two inches and a half of the transverse colon were resected, and two wounds in the mesentery were sutured, as also a nick in the duodenum. The patient died at the end of twenty hours. Union was found to be perfect at the autopsy, no leakage occurring through the sutures. There was some evident folding of the mesentery and colon from the adhesive peritonitis, which was stated to have existed in the report of the autopsy.

The following specimens are taken from the Warren Museum to illustrate certain points alluded to in this paper.

Specimen No. 919 shows a matting together of the intestines from adhesive peritonitis or from a congenital condition. The patient was sixty-five years of age, and suffered from chronic diarrhœa and disease of the kidneys. There were no symptoms of obstruction noted in the report of the case in the records of the Massachusetts General Hospital.

Specimen No. 4758½ shows the condition of the intestine of a young man who died more than a year after passing a piece of intussuscepted bowel[2] seventeen inches in length. Persistent vomiting continued afterward, and at the autopsy a constriction was found at the point of cicatrix above which the intestine was much dilated.

[1] New York Medical Journal, August 21, 1886.

[2] Boston Med. and Surg. Journal, December 28, 1876.

Such a specimen as this suggested the possible results that may follow a circular suture of the intestine, should considerable contraction of the cicatrix subsequently take place.

Conclusions.—As it was not the purpose of these experiments to test the different kinds of suture, little need be said on this point. In applying the interrupted suture it was found easy to invert a larger amount of intestinal wall than is desirable, and, as has been observed by other experimenters, there is an increased tendency to invert as the suture proceeds. Neither of these difficulties was met with in the case of resection of the human small intestine reported above. The suture appeared to be much more readily applied in this case owing to the absence of eversion of the mucous membrane, and the greater calibre of the bowel prevented undue narrowing of the lumen.

The right-angled continuous suture when applied is completely concealed and holds the edges of the wound firmly together. It has the advantage of avoiding numerous knots which tend to increase adhesive inflammation, and is drawn tighter by increased distention of the bowel. In the case of any suture, however, which inverts the coats of the intestine a valve-like action of the wound is produced, and the greater the distention of the bowel the more firmly are the edges of the wound pressed together.

In one experiment, not here recorded, the intestine was removed immediately after suture (interrupted) and forcibly distended with water, but no leakage occurred owing to this action. In applying any continuous suture, a certain amount of care and attention is required on the part of the operator to keep the stitches tight during its application. This necessitates the use of one hand solely for this purpose, which, however, does not constitute a serious objection. Moreover, failure to secure firm coaptation at any point can be remedied by applying an interrupted stitch at that spot. The continuous suture, it is rightly claimed, becomes tighter with distention of the bowel, but this supposed advantage, so far as holding the edges

of the wound is concerned, is offset by a tendency to prevent a dilatation of the wounded bowel when relief to obstruction is desirable.

The rapidity with which adhesion takes place tends to equalize the holding qualities of different sutures, they being all rapidly buried under the reparative material.

Unless strict asepsis is preserved, suture of the intestine is followed by numerous adhesions of peritoneal surfaces in the neighborhood of the wound. In some cases this is so extensive as to conceal entirely the sutured intestine, so that it is difficult to find in the mass of matted intestine. It would seem difficult for intestinal contents to pass through such a tangled coil, but in no case was there any material obstruction to the flow of fluid injected at one end. Such a condition may exist in the human intestine without symptoms of obstruction, as is shown by Specimen No. 919, Warren Museum, referred to above. Considerable interference with peristaltic action must, however, exist when adjacent coils are so closely adherent. Should a resection be contemplated at a time when obstruction is already present, as in strangulated hernia, and the indications are not only to remove the sloughing bowel, but also to relieve obstruction, it becomes a question whether intestinal suture or an artificial anus will carry the patient more safely through the crisis.

The conditions favorable for a local peritonitis are already present, and it is probable that adhesions of the adjacent portions of the intestines will, in many cases, take place as in the cases of resection of human intestine reported.

In the case of strangulated hernia the upper portion of the bowel was still greatly distended at the time of death.

When the symptoms of obstruction are the most prominent features of the case and this condition demands prompt and complete relief, it would seem preferable to establish an artificial anus and to suture the intestine later when the conditions were more favorable for recovery.

If strict asepsis has been preserved, little or no adhesion of the bowel or mesentery is seen. This is shown well in the specimen described, where there is no sign of folding or matting

of the intestine and the suture in the transparent mesentery can still be seen (page 8).

In the specimen showing the bowel three weeks after resecting, the mode of casting off of the intestinal suture was observed. The peritoneal surface of the wounds had united firmly, and union of the mucous surface was delayed from being complete by the threads of the continued suture, which, in each case, were hanging from the wound in the interior of the bowel.

This observation suggests the possibility of a delay in the union of the mucous membrane by the casting off of the long thread in the continued suture. In those operations in which only a small portion of the coat has been inverted no ridge was seen in the interior of the gut, but when the amount inverted was considerable the ridge was still observed undiminished in size at three and six months. In the suture of longitudinal wounds of the intestine, this inversion was not observed; on the contrary, a slight groove marked the line of the incision into the mucous membrane.

The inverted coats form a prominent ridge or diaphragm narrowing the calibre of the bowel, and by further contraction of the cicatrix might give rise to symptoms of stricture. Such an unfortunate sequel actually occurred in the cicatrization which followed the healing of the intestine in the case of intussusception reported above. Here we find a circular cicatrix terminating in stricture. Although under ordinary circumstances the wound would not heal in a way to lead to such an unfortunate sequel, it is well to recognize the possibility of such an occurrence if the edges of the bowel are carelessly turned in during suture and excessive local inflammation is not guarded against.

In longitudinal wounds there is also danger of too great narrowing of the bowel, not from inversion of the coats but from loss of a sufficient amount of intestinal wall. In such a case the wounds should be closed transversely instead of longitudinally, the former method resulting practically in a circular suture of the intestine.

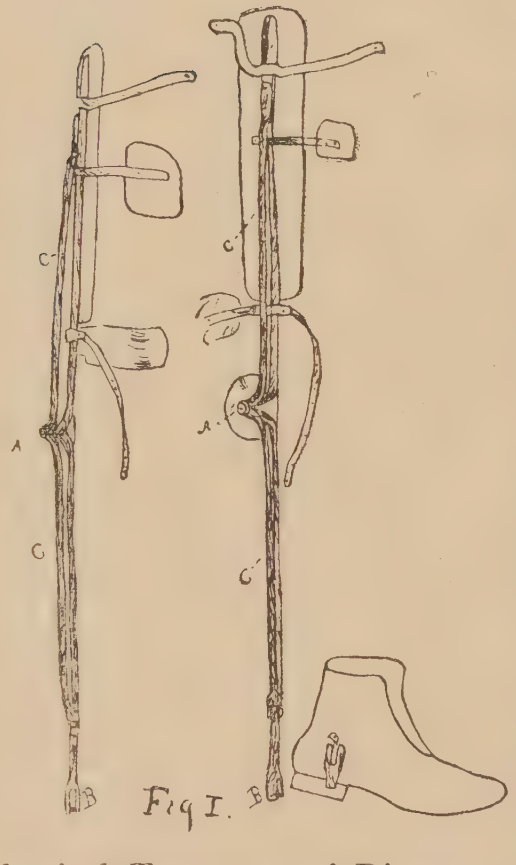
Although the peritoneal layer is an extremely thin one and forms a very insignificant portion of the intestinal wall, as seen

in microscopic section, a marked activity of cell-growth is observed on the surface of the coat and the line of sutures, within twenty-four hours probably concealed under newly formed tissue. If the bowel be removed at an early period and sections of a portion of the wound are examined microscopically, a marked thickening of the peritoneal layer will be found enclosing the cicatrix and shutting it off from the peritoneal cavity. A certain amount of cell proliferation will also be found in the other coats, both muscular and mucous. The latter seems to unite promptly, leaving little or no granulation surface exposed in the interior of the bowel. For some weeks after suture the inverted muscular layers lie parallel to one another, but are separated by a line of connective tissue. A certain amount of cell growth may be discovered in these layers, however, during this period. In the specimen examined three months after suture there was evidence of a new growth of muscular tissue in the neighborhood of the cicatrix. In the specimen examined six months after suture interlacing bands of muscular fibre indicated that completed union of the muscular wall had taken place. We found also that the submucous layer was continuous through the cicatrix as a distinct layer. A slight thickening of the peritoneal layer was also found at this period.

The different stages in the process of repair consist, therefore, in a thickening of the outer or peritoneal layer, which covers in the wound even in the mildest forms of reparative inflammation. When the reaction is stronger, adjacent layers of peritoneal tissue are called into aid in protecting the wound, all these external changes subsequently disappear, to a greater or less extent. The mucous coat unites readily, but not completely until the suture is cast off, which event probably occurs about the end of the third or fourth week in the continued suture, and much earlier in the interrupted suture. The repair of the muscular coat is slower still and is not probably complete earlier than the sixth month.

THE EXPERIMENTAL VALUE OF THE DOWSE SPLINT.[1]

BY E. G. BRACKETT, M.D.



THE value of extension by apparatus in the active stage of hip-disease is well recognized, but mechanical aid in the convalescent period has received far less attention, and its value is less appreciated, especially the splint devised by Taylor, of New York, and called by him the Dowse splint. The object of this splint was to allow the leg the motion of walking, at the same time removing the pressure from the hip-joint. It is described in his "Mechanical Treatment of Diseases of the Hip-joint," in which he says: "A strong, upright bar of steel is jointed at the knee (Fig. I, A) in such a manner, that, when vertical, the rivet of the joint is behind the vertical line. It is evident that, with such a joint, vertical weight would not only be sustained, but would add firmness to the supporting power." [2]

The lower end is connected by means of a socket-

[1] Read before the Surgical Section of the Suffolk District Medical Society, May 3, 1887.

[2] Loc. cit., p. 54.

joint (B), with a plate which is secured to the sole of the boot. At the upper, are the anterior and posterior arms for the attachment of the perineal bands, and in no way differ from the more commonly used Taylor splint. Greater strength is given by a smaller steel bar (C), parallel to the outer side of the original upright, being made a part of the joint at the knee, and extending some distance above and below, serving as a kind of buttress. The leg is kept in proper relative position to the splint by steel plates, which, in the original one, encircled both the thigh and calf, but these are now replaced, in part, at least, by simpler and less cumbersome means. The splint is so applied that the heel is raised from the ground a distance of from one to two inches, as desired, the toes being allowed to rest on the ground only after considerable flexion of the foot — a position decidedly at a disadvantage for any use. As the foot is brought to the ground, the weight of the body, resting on the perineal band, is sustained by the splint, which prevents the foot from receiving pressure. The leg serves to bring the brace into the different positions at the proper time.

When the perineal band is used to support the entire weight of the body, the parts are subjected to so much pressure, and especially, of course, with heavy subjects, that the ordinary round pad is frequently a source of continual discomfort, from concentration of so much pressure over so small an area. The same is true with less pressure, but unusually tender skin. For use in these cases, I devised a perineal band, the plan of which was to utilize all bony surface possible for support, and since it has now been used with comfort for nearly three years by a number of persons who were unable to bear the round pad, I think it has proved a usefulness.

The parts available for support are the tuberosity and ramus of the ischium, and a portion of the ramus of the pubis. The tuberosity is, of course, both in position and structure, best calculated for this work, but the ramus is also capable of considerable use. The object of the pad was to get a bearing surface as large as these bony structures would allow, and with a means to adapt this pressure as uniformly as possible to this uneven portion of the pelvis, and to change the amount in different portions of the pad. To accomplish this, three straps (A, B, C, Fig. II) pass beneath the perineum, and are firmly secured, posteriorly, to a steel bar (D), and anteriorly, to three buckles, thus allowing their length to be changed.

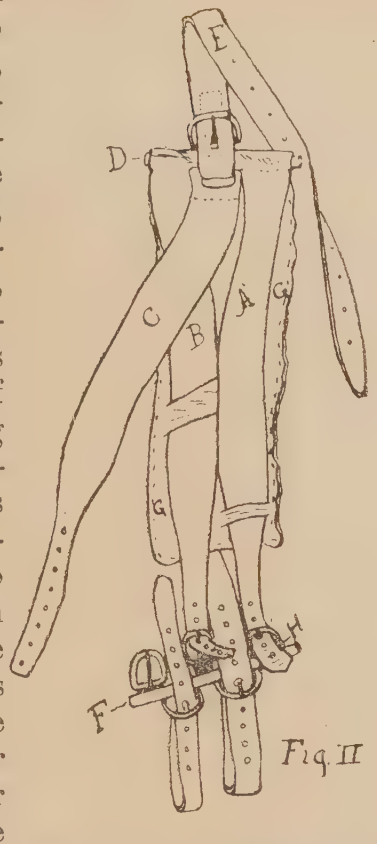


Fig. II

The posterior bar is connected by a strap (E) at its centre to the posterior arm of the brace, thus allowing either end a certain amount of vertical oscillation. The three buckles are fastened to a similar bar (F), which has two straps to connect it with the anterior arm, little or no motion being allowed. Between these straps and the perineum is a piece of leather (G), its size regulating that of the pad, which is fastened to the bar behind. This serves to transmit the pressure of the straps, and also to keep them in position, which is accomplished by button-hole slits, through which the straps pass. The position of the straps is as follows: The outer one (A) passes along the outer border, and is secured to the outer buckle (H). It should pass beneath the tuberosity of the ischium. The second, or middle one posteriorly (B), crosses obliquely inward to the inner buckle, and by this, more nearly corresponds to the direction of the ramus to which it gives its support. The third, or inner (C), crosses the one just described, and is secured to the middle buckle, and gives its special support in the space formed by the divergence of the first and second. By this crossing, the inner edge of the pad is made concave, giving better adaptation to the parts. By this arrangement with buckles, the surface can be made to fit closely all the parts serving for support. Felting one-eighth of an inch in thickness may be used to cover the leather. More than this should not be used, as it interferes with the principle of the pad.

In order to determine points of guidance in applying the pad, that it should occupy this relative position to the bony parts, I took measurements of different pelves, with the following result: The rami diverge in a horizontal plane, at an angle which, in the average of those measured, was fifty-one and one-half degrees, and this line of direction, carried backward and upward on the dorsal surface of the ilium to the level of the anterior superior spine is, at this point in the female, two-thirds, and in the male, a little more, the

distance from the anterior superior spine to the middle of the sacrum. Therefore, a line drawn from this point to the symphysis pubis would pass over the tuberosity and ramus. That the pad should have perfect apposition to this surface, it must pass over this obliquely. and it must be so applied, that posteriorly, its outer border shall be within this line of direction, and anteriorly, the inner border shall be on the outer side. In this position, the straps will come beneath the respective parts, as described.

The time for which Taylor intended the use of the splint was "after all pain and sensitiveness in the joint have ceased, and when there is no longer a disposition of the muscles to take on abnormal contraction, and there is free motion in the joint," for, he says, "the motions of the joint may be kept up with only good, and no harm, so long as pressure is removed, till the joint is completely well." [3]

This stage is certainly a trying one. In the absence of the inflammatory condition, the patient no longer is impelled to keep the joint at rest, and there is yet to be a long period of time occupied in repair, during which the articular surfaces are not capable of withstanding the effect consequent on a return of the use of the leg, and an important problem which now presents itself is: how can the patient be given the greatest freedom, and, at the same time, the joint be protected from any influence which may retard the process of repair.

While motion at this time is, of itself, not injurious, and certainly of much value to the long disused muscles, the recently inflamed structures are in no way capable of withstanding the combined effect of motion and pressure. Therefore, we must, in some way, prevent the joint from receiving the pressure incident in ordinary walking. Crutches and a high shoe remove pressure and protect the joint from injury, provided no accidental one comes to the leg while allowed to freely swing; but there are many cases, especially in older subjects, with whom there is urgent demand for the release from all possible restraint during this period of repair. If motion alone is not harmful, then any means which permits the leg the freedom in the movements of walking, but, at the same time, relieves it of pressure, will allow the joint the condition necessary for its repair, while the convenience to the patient is of the greatest; and if the Dowse splint accomplishes this, its theoretical use is correct.

Of the value of any appliance, we have two kinds

[3] Loc. cit., p. 53.

of evidence, clinical and experimental. Of clinical, experience has proved this splint to be a valuable means of treatment in this stage of the disease, and in illustration, I will briefly allude to a few cases in which this splint has been used, and for the opportunity of presenting two of these, I would acknowledge my indebtedness to Dr. E. H. Bradford.

As it is not my object to report the cases, but only to use them to exemplify this treatment, I shall not resort to details of the history, but state conditions.

Case I. Girl, six years of age. Three years ago was seen early in the acute stage of hip-disease, and at once given a Taylor hip-splint and crutches, with extension at night. When signs of inflammation had disappeared, the splint was connected with the shoe, in the same manner as is the Dowse, but no motion at the knee was given. By this difference doubtless the pressure on the toe is less than when motion at the knee is given. She has worn this about two years, and her improvement has been steady. There is now no difference in appearance of the two limbs; there is free motion in about 40°, and without the splint there is a scarcely perceptible limp.

Case II. Girl, fifteen years of age. When eleven, came under treatment for acute hip-disease, and after rest in bed for two months, was given a Taylor hip-splint and crutches, which were used for two years. At this time there was free motion in about 35°, and there was no pain or tenderness. Pressure gave no pain, but the leg was able to give but little support when the attempt was made to walk. The Dowse was given, and with this she was able to walk. For three months she kept under observation and continued to do well, but was then not seen for fifteen months. At this time she was in excellent health. The two limbs were nearly equal in size and strength, the amount of shortening was the same as when measured for the Dowse. It was found that after wearing the splint for a year, she gradually gave up its use, and has since walked without mechanical aid.

Case III. Male, twenty-six. At the age of fifteen there was disease about the hip, with abscess formation. When twenty-three he considered himself well, and was doing hard physical work under which, periarticular disease appeared, but involved the joint enough to cause the usual symptoms of disease of the head of the bone, as pain, muscular spasm, inability to use the leg. The sinus re-opened and discharged for a while. Under rest, these symptoms subsided, and he was then put on the Dowse, and with the aid

of a cane was able to go about with ease. He has now used this for a year, and the improvement in the condition of the limb has been constant and satisfactory.

Case IV. Male, twenty-seven. Nearly six years ago had acute articular disease. After some months in bed, a Taylor splint and crutches were used for two years. After this treatment there were no longer signs of inflammation: there was free motion in about 30°. Pressure gave no pain, but it was only with considerable effort that the leg could be used to support the body. A Dowse splint was given, and has been worn from that time. There has been no interruption in the improvement of the limb, until at present it offers no restriction to the work which ordinarily would come to any one.

Although we have evidence of the clinical value of this apparatus, yet that it accomplishes the work which is claimed, has been doubted by some. In order to have an experimental estimate as well as clinical, of its value, I have endeavored to ascertain the actual work accomplished by it, and I am indebted to Dr. Bowditch, of the Medical School, for the opportunity of attempting these experiments, and also for his advice in the work.

As the Dowse splint is applicable only to those cases in which, as far as possible, the motions of natural locomotion are to be allowed, protecting the joint from pressure while admitting motion, its application must be to the mechanism of walking, and its adaptation such as to protect the joint, in the direction, and at the time of greatest shock and strain. Therefore the different motions of the step must be separated, in order to determine the time and direction of force from which by the apparatus we protect the joint. The division of the step into the different positions, is now accurately accomplished by instantaneous photography, and in the study of these I have used the series taken by Mr. Eadweard Muybridge, in 1881, and from which the figures in Plate III, are copied, by permission. Only such as have relation to the subject will be considered.

The chief element in the progression is the successively falling forward of the body, swinging on the ankle as a pivot, in each step being raised to allow it to fall again, or as Holmes has expressed it, "a perpetual falling, with a perpetual self-recovery." Therefore the body describes a series of regular curves. which correspond to the vertical oscillation, the amount of which is influenced by the length of the step. The

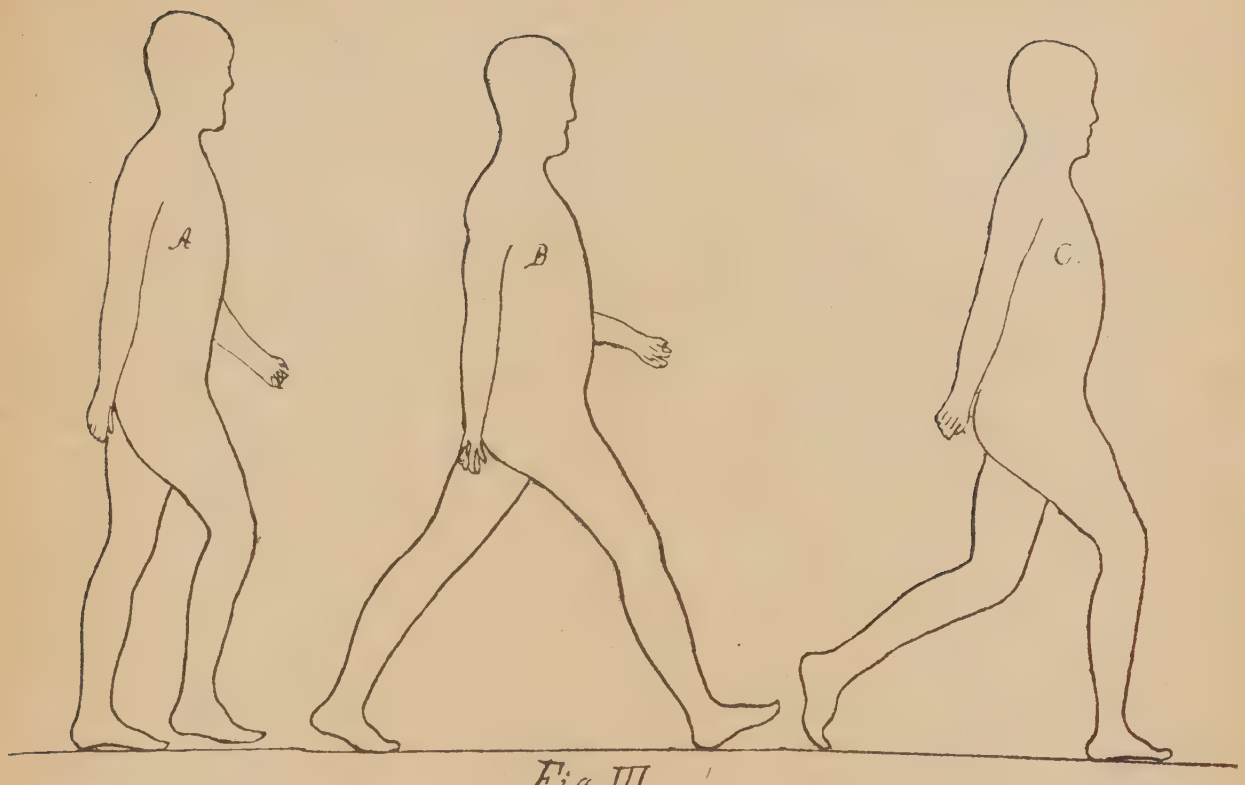


Fig. III.

body reaches its highest point in this vertical curve, at a time when it has just passed a point directly over the supporting foot. From this time it falls forward, until arrested by the foot which has been placed in advance and as the body passes on over this, it is raised by muscular effort to the summit of its curve. It is evident that the force exerted by the leg is by no means a uniformly distributed one, and it is the relative amount of strain, and the position in different parts of the step, that we have to study in dealing with this subject. When the body is in the summit of its vertical curve, the supporting, or active leg, is straight, (Fig. III, A), the other, or passive leg, is taking advantage of this moment of greatest altitude of the pelvis, to swing forward, with the knee flexed, to its position in advance. This leg remains flexed until the thigh is at that angle with the trunk, in which it is to receive the weight of the body. The foot continues to swing forward, until the leg becomes straight, and in this position the leg is held until it is brought to the ground by the falling forward of the body. (Fig. III, B). In this way the heel is the first to strike and receive the greater part of the shock, which comes to the foot at this time. The toe is soon brought to bear its share of the pressure in a time equal to a little more than one-sixth of the whole im-

pact. Should the leg remain thus extended, this position would represent the body in the lowest point in its vertical oscillation, and its entrance on the ascending portion of the next curve, would be sudden, changing its direction at an angle. But at this moment the knee is slightly flexed, and by the shortening of this support, allows a continuation of the downward motion ; thus its gradual checking, and the lowest point in the vertical oscillation is after the moment the heel strikes the ground. This position at this moment is represented in Fig. III, C. The foot which is about to leave the ground rests on the toe, while the supporting foot is flat on the ground, the leg bent at the knee. Carlet found this lowest point to occur at the middle period of double impact.[4]

As the body passes onward, it is raised to the summit of its curve by the straightening of the leg, which is complete at the moment the body has passed a point above the foot. (Fig. III, A). The other foot has now left the ground, and is swinging forward to take its place in advance. The manner in which it leaves the ground is as follows : As the body passes onward, the heel is raised, at the same time the knee flexed, allowing the toes to rest alone, (Fig. III, B and C), until the distance becomes greater than the length of the leg, when it is raised from the ground, and with the knee flexed swings forward. The body is now ready to again fall forward, and the process is repeated. Thus we can divide a step, for our subject, into three phases :

First, would extend from the highest point in the vertical oscillation, to the moment the heel strikes the ground, or the period of descent; Second, from the time the heel strikes the ground, until the lowest point in the vertical oscillation is reached, or the period occupied in checking the downward motion ; and Third, from this last, to the time when the body reaches the highest point in the vertical oscillation, or the period of ascent. It must appear that the pressure is far from uniformly distributed over the two parts of the foot.

As the foot first strikes the ground, the centre of gravity is behind the ankle-joint, and the heel alone receives the shock, and sustains all weight for a time, when the toe is brought to the ground. Then, as the body passes to a point over, and then in front of the foot, the heel bears less, and the toe more, until the heel is raised, and the toe, for a moment, rests alone.

With a view of determining the estimate of this

[4] Annales des Sciences Naturales, xvi, vi, 54.

pressure, I performed a series of experiments. The apparatus used was, in principle, the same as used by Carlet, and consisted of thick rubber cylinders, with air-chambers connected with Marey drums. These rubbers were fastened to the shoe, one under the heel, and one under the toe, and the drums were so regulated that the amount of excursion of the needle which would be caused by the weight of the body was known in each. This difference is shown in the following tracings, which are of the heel and toe, taken in the same step (Fig. IV). As these are examples from the walk of different individuals, they naturally show some points of difference, such as characterize an individual gait, but all show the distinctive features for which they were taken. The amount of pressure is indicated by the elevation of the curve, while the dotted line represents the height which corresponds to the weight of the body. As is seen, the heel receives alone all the pressure which comes suddenly to the foot, and that it is only after a well-marked interval that the toe takes an active part. As the centre of gravity of the body passes in front of the ankle-joint, the pressure is gradually transferred to the toe, and for a moment, at the close of the impact, all pressure is sustained by this part of the foot.

There are two parts of each impact, where, theoretically, more pressure comes to the foot, than that which represents the weight of the body. In the second phase, when the descent of the body is checked, there must be used the additional force necessary to check a weight equal to that of the body falling a distance of the vertical oscillation, which is, according to Carlet, about fourteen millimeters in an average step.[5] Also in the third phase, when the body is raised to the highest point, there will be this corresponding additional force required. Carlet considered this additional force represented a weight not exceeding one-fifth that of the body.[6]

[5] Animal Mechanism, Marey, p. 118.
[6] Loc. cit., p. 25.

In combining the two parts of these tracings to represent the total pressure on the foot, I obtained a curve in the first part of the impact, above the line of body's weight, indicating an additional amount at this time. In the latter portion, the tracing remains at, or very near, the level of the dotted line, showing that at this time, when the body is falling forward, the force used is only that necessary to keep the body at a fixed distance from its pivot. Therefore, there is nothing in the pressure-curves of ordinary walking indicating the push of the toe so often mentioned. A glance at the photographs will show that such force applied at this moment would not give a forward motion, but would only raise the body in a nearly vertical direction, while it is, at this time, scarcely at the lowest point in the downward direction.

In regard to position, we find that in the beginning of the impact, the leg is extended, and the heel receives alone the shock. The leg is immediately slightly flexed, and the toe is brought to the ground after an interval of about one-sixth of the time of entire impact. Then, with the foot flat on the ground, the leg is straightened, and the body raised. As it now falls forward, the heel leaves the ground, and, for a short interval, the toe rests alone. It is an important fact that, at this moment, there is no call for extra expenditure of force, and also, there is no motion in the hip-joint.

But, in substituting a mechanical means as a part of so complicated and well-balanced a mechanism as that of walking, necessarily there results a change in the detail of its performance, and such does occur in walking with the aid of the Dowse splint. In ordinary walking, all pressure sustained by the ankle-joint is distributed to the foot, influenced by the relative position of the body and the time of impact. As both parts of the shoe are securely connected with the upright portion of the splint, so it is the object that the pressure on the shoe should be transmitted to the splint, and the foot prevented from taking part in this by its elevation. The splint should not only receive the weight of the body in the beginning of impact, but retain it until the foot is about to leave the ground. To accomplish this, the splint must be held straight during this time, and as this occurs at that time when, in natural walking, the knee is flexed and then extended, a change from ordinary walking must take place. To prove the occurrence of this change from the normal walking, I photographed, while in motion, an adult wearing a Dowse. The positions in this part

Fig V

of the step may be seen in the accompanying photographs. As the foot swings forward, the splint becomes straight, as does the leg in natural walking, and in this position, receives the weight of the body (Fig. V, A), but at this moment does not bend, as does the knee, as has been described, but remains straight, supporting securely the weight of the body. Therefore, the body, instead of reaching the highest point in its next vertical oscillation by a direct lift, must swing upward through an arc of the circle, which has the splint for its radius. The positions during this movement are seen in Fig. V, B and C.

A jar to the body from this sudden change in direction is prevented by the elasticity of the brace. Passing this point, the body now falls forward, held by the splint, until, just before the termination of the impact, when the heel is raised, and the splint can no longer receive all pressure, as before.

Thus the foot is protected from pressure, except in the last moment, when it is in the act of leaving the ground, and this at a time when the least is required.

Since the toe is allowed a position in which it may momentarily bear pressure, necessarily the opportunity

is taken, and a certain amount is sustained by this part of the foot. To determine this, I took a series of tracings in the same manner as with natural walking (Fig. VI). In these, the lower curve represents the pressure sustained by the splint, the upper, that by the toe. It is seen that the brace receives the weight alone for the greater part of the step, and in that part of most strain, while that of the toe is but of short duration, is gradually imposed, and, in amount, is never great.

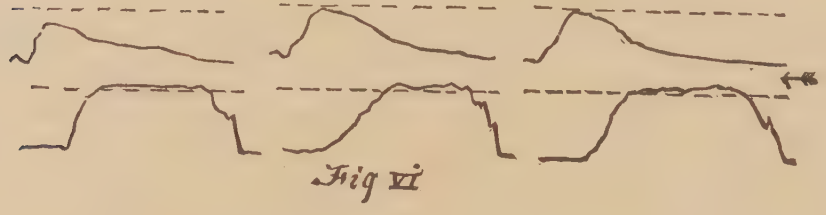


Fig VI

Therefore, of its work I think we can say that, in the first portions of the impact, it protects the leg from pressure, and this at a time when greatest strain is usually sustained by the leg. That slight pressure is allowed the toe for a moment at the close of the impact, but this comes at a time when motion is not occurring in the hip-joint. Knowing the actual protective value, in addition to the clinical evidence of its usefulness, we need no further explanation for its application, except the conditions under which we wish the joint to be while in its treatment. It must appear that, in the proper cases, it is a valuable means of treatment. Certainly, there is a time in the convalescence when the joint can bear this amount of pressure, as has been described; yet the weight during the whole step, and especially in the first of the impact, when there is considerable shock to the joint, could in no way be allowed with impunity. This being removed, but little actual work is left for the leg to do. However, more intelligent care on the part of the patient than with ordinary apparatus is necessary, for the opportunity is present to make more use of the limb than is intended. Unless the splint is straight, as has been described, it does not bear the entire weight.

There is no reason why its use should be restricted to hip-disease, but it may be employed in any case where the leg is to be relieved of its work, or a portion of its work; and Taylor recommended it for permanent use after exsection of the head of the femur, and in ununited fracture of the neck of the femur, there is certainly opportunity for experiment.

FRACTURE OF THE SPINE: ITS IMMEDIATE TREATMENT BY RECTIFICATION OF THE DEFORMITY AND FIXATION BY PLASTER OF PARIS JACKET.

By HERBERT L. BURRELL, M.D.

Surgeon to Carney Hospital; Surgeon to Out-Patients Boston City Hospital; Ass't. Surgeon Boston Children's Hospital; Demonstrator of Bandaging and Apparatus in Harvard University.

Read at the Annual Meeting of the Massachusetts Medical Society, June 7, 1887.

FRACTURE OF THE SPINE:

Its Immediate Treatment by Rectification of the Deformity and Fixation by Plaster of Paris Jacket.

From a practical point of view, we may consider dislocations and fractures of the spine together, and it will not be a difficult task to represent to the minds of most surgeons the utter hopelessness of this injury in most cases.

All authorities[1] agree that the prognosis depends largely upon the location of the injury, and the amount of damage done to the spinal cord. It is not the injury to the bony structures or ligamentous union, that renders this injury so fatal, but the pressure or crushing that takes place of that wonderfully constructed spinal cord, which receives and transmits impressions to the various members of the body.

The spine itself is so complexly constructed, adapted to so many different movements and purposes, that we can really say, that when it receives an injury, the "back bone" of the human organism is broken.

The cord resting as it does in a bony canal, may be pressed upon by bony spiculæ from any side, it may be pressed upon or severed completely by some sharp fragment, or simply the anterior buttress or body of the vertebræ may be crushed upon itself, without any serious or permanent

[1] Sir Astley Cooper, Fractures and Dislocations of Joints; Malgaigne, Fractures and Dislocations; A. Shaw-Holmes, System of Surgery; Gurlt, Handbuch der Lehre von dem Knochenbrüchen, Hamm. 1864; Sir Charles Bell, Observations on Injuries of the Spine and of the Thigh Bone, 1824; Hamilton on Fractures and Dislocations; Bryant, Practice of Surgery.

injury to the cord having occurred. This is illustrated in Plate X.

The higher the injury of the bony column, the greater fatality. Gurlt[2] reports that out of 178 cases where the cervical vertebræ were fractured, death occurred in 164, or $90\frac{1}{7}\%$. Out of 184 in the dorsal region, death occurred in 146, or $79\frac{2}{3}\%$. Out of 82 in the lumbar region, death occurred in 56, or $68\frac{1}{5}\%$. This ratio of mortality is quite what we should expect, as the nearer we approach the respiratory centre, the more fatal is the injury.[3]

By the courtesy of the Surgeons of the Boston City Hospital, I have been enabled to collect all the cases (82) that have occurred in that Hospital, and have tabulated them, to ascertain what facts they will show The data thus obtained is represented in Tables D, E, F, G, and H, and the lines represented opposite present a synopsis of the detailed account that appears in the tables.

First, regarding the mortality of these cases, we find that the fatality is very great, and this is represented by Table A 1.

The fatality I have further analyzed to show mortality according to location. This is represented in Table A 2.

The striking immediate fatality of the accident is shown in Table A 3.

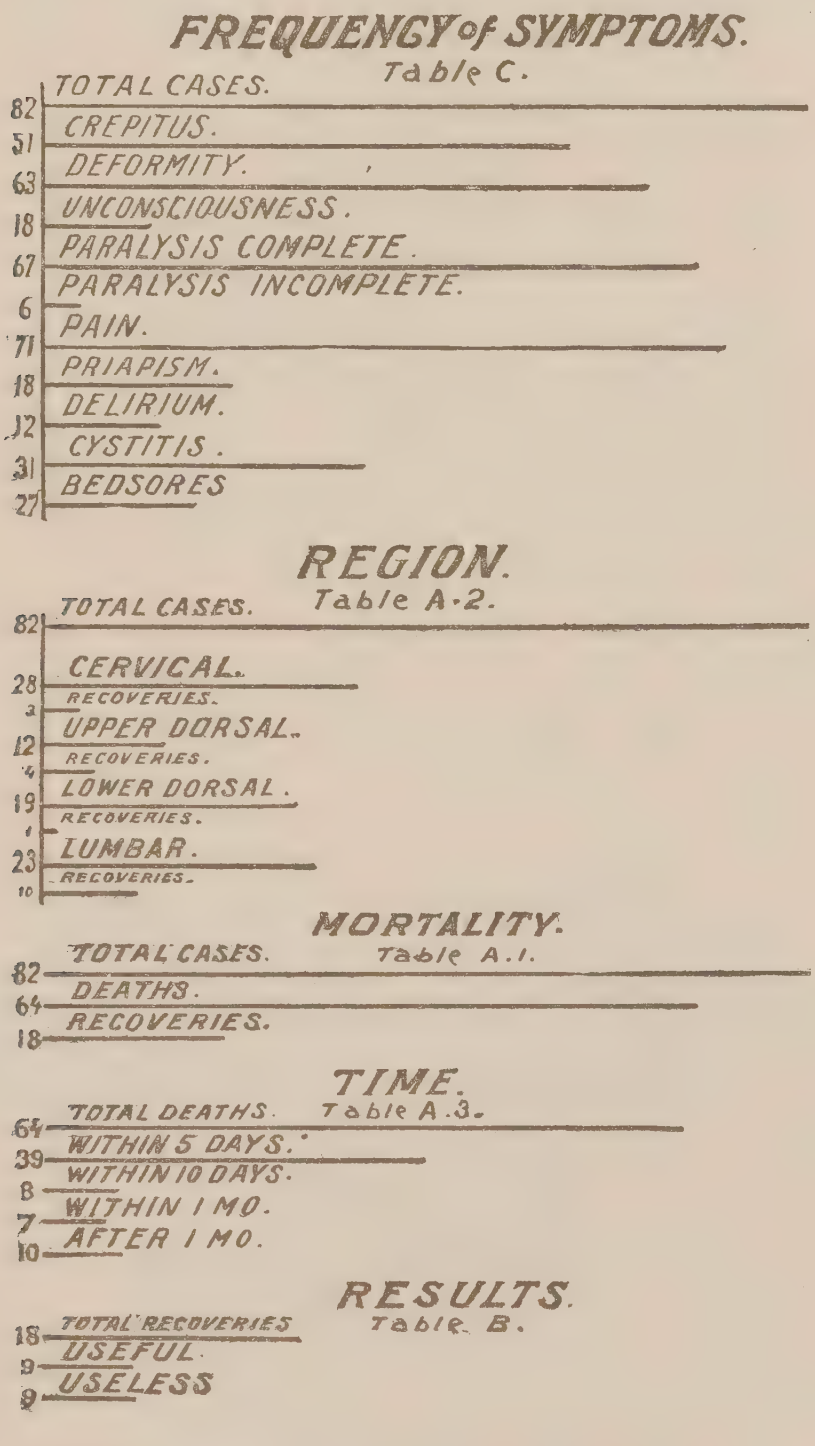
The recoveries have been divided into two classes:—the first, where the patient was useful, in the sense of being self-supporting; the second, useless, where the patient is bed-ridden, and unable to earn a livelihood. So that we really have in these 82 cases of Fracture of the Spine, an apparent recovery of 22%, where really there is only 11% returned as producers in the community. This is shown in Table B.

The ratio of frequency of prominent symptoms occurring after fractures of the spine, is shown in Table C.

[2] Ibid. p. 72.

[3] Bryant, ibid. p. 105.

The utter hopelessness which is expressed in the term broken back, pervades the minds of all practitioners, and



perhaps Erichsen[4] expresses the general feeling, when he says that "Fractures of the Spine through the bodies of the vertebræ with displacement, are inevitably fatal."

[4] System of Surgery.

The treatment of fractures of the spine may be divided into three principal heads :—

a. Expectant. Water bed. Air bed. Wire bed. Bonnet's vertebral gutter. Extension and counter-extension.

b. Operative. Trephining. Removal of bony fragments.

c. Rectification of the deformity and fixation of the spine by plaster of Paris jacket or other apparatus.

a. EXPECTANT.—The statements made by Cline[5] and by Cooper[6] that we can accurately determine whether the body, or the arch, or the spine of a vertebra is broken, is not supported by facts. Further, Cline and Cooper believed that death was inevitable, sooner or later, if the fragment were not lifted by an operation.

This is possibly true, but specimens like Plate I. and Plate II. show only too clearly that, at times, we have to do with an irremediable injury, and our duty in such cases is to pursue the expectant plan of treatment—that is, placing the patient on an air or water bed, and treating the symptoms as they arise. By this means we can prolong life, and make existence bearable. It is possible that a certain amount of relief may be obtained in these severest cases by permanent extension and counter-extension.

Having decided that the expectant plan of treatment must be pursued in certain rare cases, we come to our second division, that of

b. OPERATIVE.—What further can I say to you than has been said in that bitter controversy between Sir Charles Bell[7] and Sir Astley Cooper, as to the expediency of operations on the spine. Only on the parts posterior to the spinal cord, could an operation for a moment be entertained; for to

[5] Chelius Surgery, Vol. 1, p. 590.
[6] Sir Astley Cooper on Disl. and Fract., 1851, p. 479.
[7] Observ. on Injury of the Spine and of the Thigh Bone.

attempt to remove the body of a vertebra after fracture of the spine from behind, would necessitate the division of the spinal cord; to attack it in front would be equally inad-

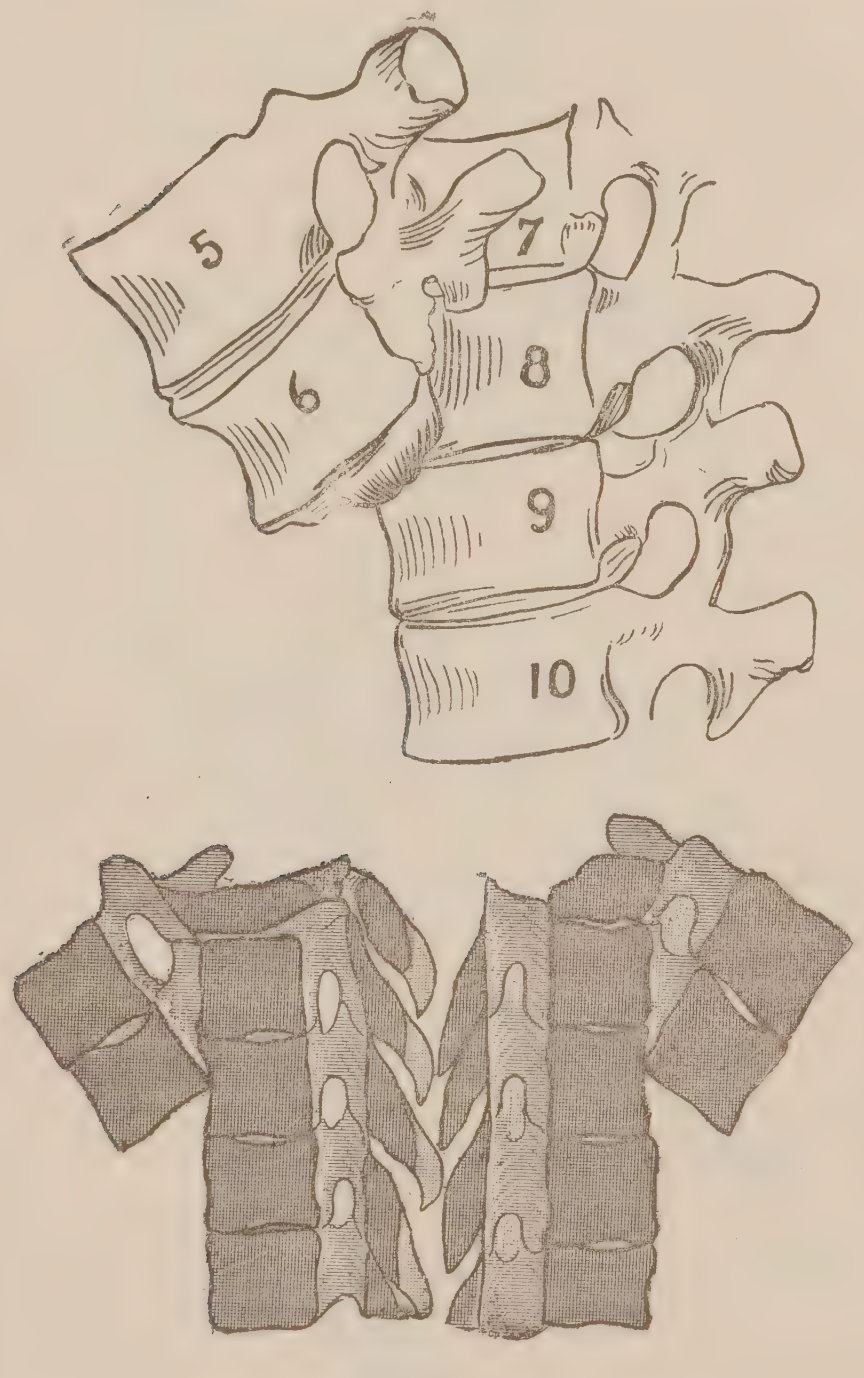


PLATE I.

(No. 1229 WARREN MUSEUM.)

The patient fell 30 feet. Wild and irritable, complete paralysis, bed sores, cystitis and death from exhaustion in two months. There has been a complete rupture of the intervertebral substance between the 6th and 7th vertebræ, the upper edge of the 7th being carried away to the right side with the 5th and 6th vertebræ. The state of the spinal cord in this case may be imagined.

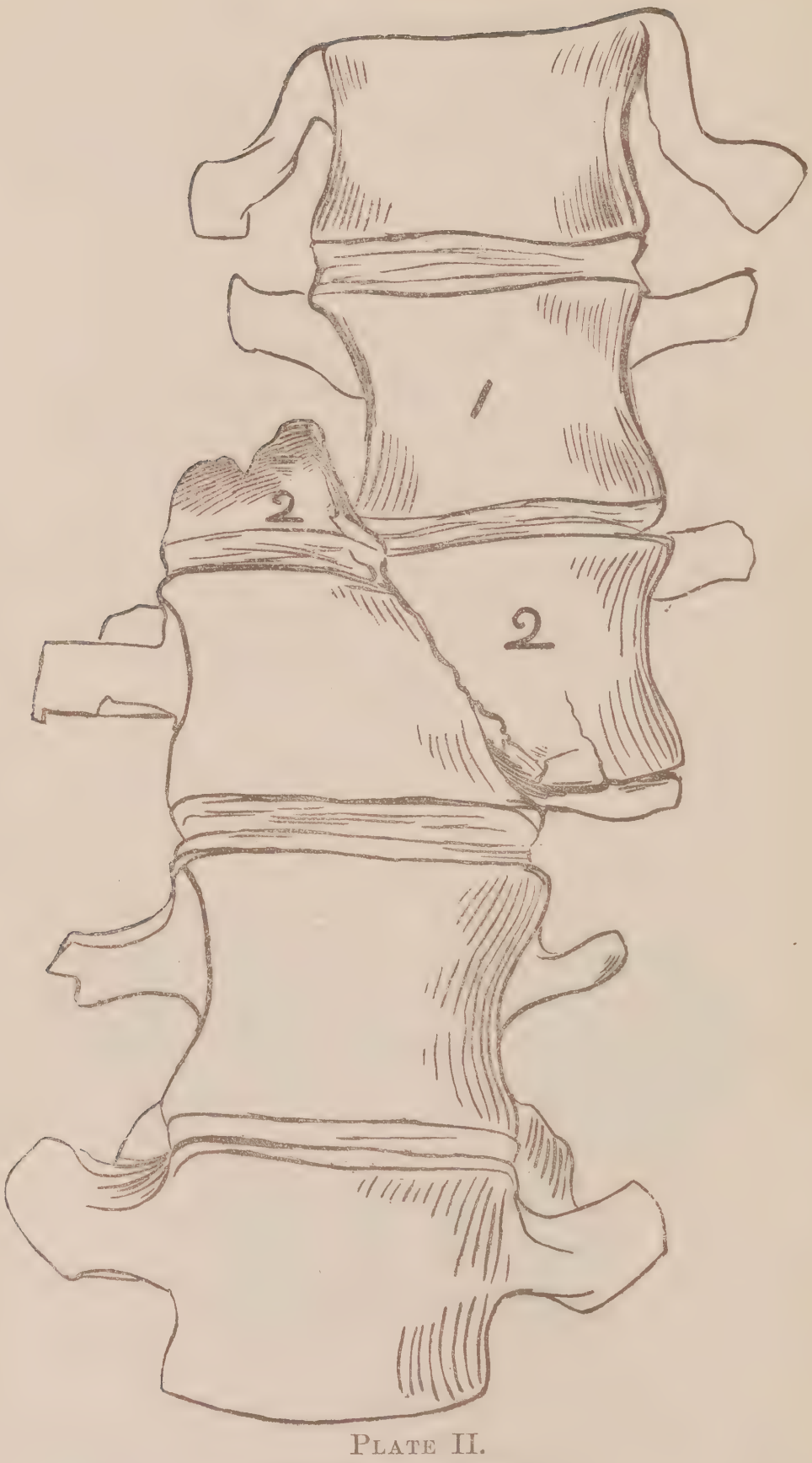


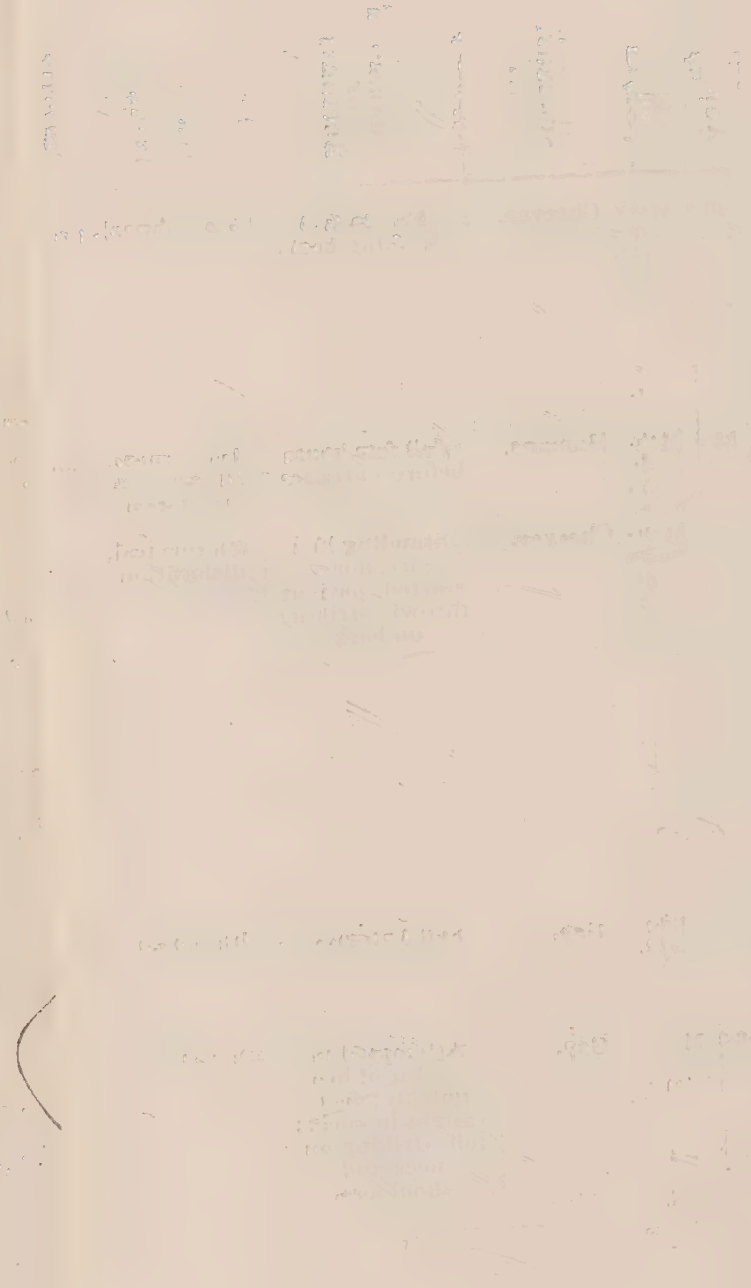
PLATE II.

(NO. 139 WARREN MUSEUM.)

T., æt. 19, run over by a fire engine. Complete paraysis, bed sores, cystitis, and died of exhaustion. There is a very extensive fracture and displacement of the 1st and 2d lumbar vertebræ. Cord completely destroyed.

(Table D.) TABLE OF OPERATIONS.

Number.	Name. Date. Reference.	Age.	Sex.	Surgeon.	Character of Accident.	Seat of Injury.	Crepitus.	Deformity.	Unconsciousness.	Paralysis.	Pain.	Priapism.	Delirium.	Cystitis.	Bed Sores.	Deaths.	Autopsy.	Remarks.
21	J. G. Jan. 14, 1867. vol. 9, p. 200.	40	M	Cheever.	Fell 25 feet, striking back.	5 & 6 dorsal.	yes	yes		Complete.	yes	yes	no	no	no	yes, next day.	no	An incision six in. long through muscles to spine. The spinous processes of 2, 3, 4, 5. d. vert. were found broken off, and were easily removed. Portions of laminæ of 3 and 4 removed with trephine and "rongeur" forceps. All spiculæ and sharp points removed. Cord exposed from 2 to 5 d. vert.; opposite the 3d, slight laceration of cord. Emphysema was now first noticed on r. side, and soon covered it. Pulse and respiration better than at beginning of operation. Pupils contracted; surface warm; cold compress on wound. Conscious that night; took nourishment well. Paralysis continued. Urine did not flow until pressure was applied: priapism nearly gone. Comfortable until noon of next day, when dyspnœa became marked. Became unconscious; died at 2 P.M.
22	J. G. Aug. 6, 1867, vol. 14, p. 156.	33	M	Homans.	Fell few hours before entrance	Tenderness, 7th cervical, 1st dorsal.	no	no	no	Complete below nipple.	yes	no	no	no	no	yes, 48 hrs.	no	An incision over 1 and 2 d., and 7 cerv. vert. was made. Some abnormal mobility was found, but no crepitus.
23	R. S. Dec. 14, 1867, vol. 13, p. 186.	36	M	Cheever.	Standing in a cart, horse started, patient thrown, striking on back.	6th cervical, dislocation.	no	yes	no	Complete of legs; partial of upper extremities.	yes	no	no	no	no	yes, within 24 hrs.	yes	9 P.M. same day, Dr. C. saw patient, and found cavity at nape of neck. Just above 7 cervical, spinous processes of 4, 5, 6 cerv. vert. could not be felt. Patient could rotate head, but pain over 7 cerv. vert. Decided to wait until morning. On A.M. following, Dr. C. made incision 3 in. long, extending from 7 cervical upward, without ether. 6 cerv. vert. found dislocated forward; no force could bring it into place. Spinous process and r. lamina and part of left lamina of 6th vert. and part of lamina of 5th removed by trephine; cord laid bare for 1 in. in length and nearly its whole breadth; membrane not ruptured. During operation, slight pressure was accidentally made on the cord; immediately patient would show signs of severe pain; pulse was slowed, and became intermittent; the moment pressure was removed, the pulse returned to its former condition. Application of cold produced the same effect. Little blood lost, and strength good: brandy twice. From this he failed rapidly: temp. 110° in axilla at 5 P.M.; and died 6 P.M. *Autopsy.*—The 6 cerv. vert. was dislocated forward, its articular processes being in front of those of the 7th. No fracture of bone, or laceration of ligaments; an abrupt depression in spinal canal at point of dislocation. Post. lig. stretched tightly over upper margin of 7 cerv. vert. Cord and membranes normal to the eye.
70	J. C. Aug. 20, 1884, vol. 121, p. 175.	28	M	Gay.	Fell 3 stories.	4th dorsal.	yes	yes	yes, at first.	Complete below 5th rib	yes	no				4th day.	no	An incision was made over spines of upper dorsal vert. Spinous process of 4th was found to be broken off, its body tilted and dislocated inwards, the articular processes of the vertebra visible. Extension, movements of neck and body, and traction by forceps, all failed to accomplish replacement.
71	S. G. Dec. 28, 1884, vol. 126, p. 72.	53	M	Gay.	Attempted to get out of bed quickly; feet caught in sheet; fell, striking on neck and shoulders.	5th cervical.	yes	yes	no	Complete.	yes					In 36 hrs.	no	Dr. Gay cut down on to the deformity, but failed to reduce it.



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(Table E.) TABLE OF AUTOPSIES.

Number.	Name. Date. Reference.	Age.	Sex.	Surgeon.	Character of Accident.	Seat of Injury.	Crepitus.	Deformity.	Unconsciousness.	Paralysis.	Pain.	Priapism.	Delirium.	Cystitis.	Bed Sores.	Death.	Autopsy.	REMARKS.
23																		(See Table of Operations.)
28	J. W. Aug. 29, 1870, vol. 30, p. 138.	22	M	Thorndike.	Fell from staging on back.	Tenderness, 7th cervical.	no	no	no	Complete.	yes	yes				4 days		Abdominal breathing; consciousness in special senses preserved to the last. *Autopsy.*—Heart, lungs, spleen, liver, kidneys, congested; 5th cerv. vert. dislocated forward ¼ in.; spinous process sunk downward nearly ½ in. from spinous process of 6th vert.; cord considerably softened from 3d to 7th cerv. vert.; effusion of blood under dura mater, at level of 7th cervical and 1st and 2d dorsal nerves, but not enough to compress cord or nerves. The canal was narrowed from 1-8 to 3-16 of an inch.
33	P. C. Aug. 31, 1872, vol. 43, p. 118.	30	M	Gay.	Fell 5 days before entrance, striking on back of neck.	Cervical dislocation.	no	no	no	Complete below nipples	yes			yes		7 days	yes	*Autopsy* showed 5th and 6th cerv. vert. dislocated; softening of cord corresponding to seat of injury.
35	C. M. March 23, 1873, vol. 45, p. 230.	45	M	Thorndike.		4th & 5th cervical.	yes	yes	no	Complete below nipples	yes	yes				next day	yes	Attempts were made in this case to reduce the deformity, without avail. *Autopsy*—Examinations of organs not remarkable; 3, 4, & 5 cerv. vert. fractured; cord pressed upon and softened. On pressure upon these vertebræ, post mortem, the deformity could be reduced, and pressure of cord relieved.
43	J. B. Sept. 10, 1875, vol. 61, p. 3.	67	M	Gay.	Fell quite a distance; did not enter hospital for 6 days.	5th cervical.	no	slight		Complete.	yes	no	yes	yes	yes	8 days	yes	*Autopsy.*—Granular kidney; cystitis; left pleuritis with effusion; slight lepto-meningitis. There was a fracture of r. transverse process, and part of the body of the 5th cerv. vert., with injury of the inter-vertebral substance.
51	C. E. Sept. 6, 1879, vol. 84, p. 240.	39	M	Fifield.	Struck on head, and knocked to ground.	Dislocation of atlas.	no	yes	yes	Complete.	yes					28 hours	yes	Extreme perspiration and cyanosis. *Autopsy.*—Dislocation of atlas, with rupture of lateral ligaments.
54	W. P. Sept. 26, 1880, vol. 92, p. 24.	52	M	Fifield.	Struck by a railroad train.	Multiple injuries; fracture of 2 or 3 vert. in dorso-lumbar region.	yes	yes	no	No.	yes	no	no			2d day	yes	*Autopsy.*—There was a fracture of 2 or 3 ribs near the dorso-lumbar region. The spine itself was broken transversely through the body of the first lumbar vert., and a segment of the last dorsal vert. was broken off.
57	W. W. Aug. 12, 1881, vol. 98, p. 87.	45	M	Fifield.	While drunk, fell 15 feet.	7th dorsal.	yes	yes	no	Complete.	yes			yes	yes	in 119 days	yes	Next day after entrance, sense of constriction about waist. *Autopsy.*—Pleuritis with effusion; emphysema of lungs; cystitis; pyelitis; fatty liver; fracture of anterior part of 7th dorsal, with compression, and complete disintegration of cord.
62	S. D. June 3, 1882, vol. 103, p. 164.	83	M	Ingalls.	Fell 18 feet.	4th cervical.	yes	yes	semi	Complete.	yes		no	no	no	next day	yes	*Autopsy.*—Fracture of body of 4th with dislocation forward; fracture of transverse processes of the 3d and 4th cerv. vert. Complete disorganization of the cord.
59	H. B. W. March 22, 1882, vol. 101, p. 94.	68	M	Ingalls.	Fell 15 feet, striking on back.	7th to 12th dorsal.	yes	yes	no	Complete.	yes		no	no	no	in 12 hours	yes	Apparent depression at 7th and 9th dorsal, and dislocation, with prominence of the 11th and 12th dorsal, with deflection to the left. Extension to legs; counter-extension by raising foot of the bed. *Autopsy.*—Fracture of the 12th dorsal, with part of the body of 11th: compression of the cord; fractured rib; pyelo-nephritis.
63	T. T. June 20, 1882, vol. 103, p. 254.	27	M	Ingalls.	Fell 15 feet.	6th cervical.	no	no		Complete.	yes	yes		yes	yes	in 69 days	yes	Tympanitis. *Autopsy.*—Fracture of the body (transverse) and articular processes of the 6th cerv. vert.; amyloid degeneration of spleen, liver and kidneys.
73	M. C. May 27, 1885, vol. 128, p. 176.	44	F	Homans.	Fell down stairs.	8th dorsal.	no	no	no	Complete.	yes		yes	yes	yes	in 3 mos.	yes	*Autopsy.*—Fracture of 8th dorsal, with crushing and displacement backwards; traumatic myelitis at seat of fracture. Spinal cord at seat of injury reduced to ½ the size, and on section it was found to consist of a tube, the wall formed by pia, with a small amount of nervous substance adherent, and a cavity the size of a lead pencil, filled with a thin puriform fluid. The cord, for about 1 cm. above and below the above site, was softened, yellow and opaque, there being no distinction between the gray and white portions. At this point, the 8th dorsal was pressed backward so as to reduce the spinal canal one half.
80	G. F. Nov. 24, 1886, vol. 181, p. 125.	74	M	Cheever.	Fell 30 feet.	12th dorsal.	yes	yes	semi	Complete.						in 18 hours	yes	*Autopsy.*—Fracture vertebra; red softening of cord; hemorrhage into pia, plural cavities, lung and mediastinum.
78	H. S. Aug. 10, 1886, vol. 138, p. 136.	45	F	Burrell.	Fell 10 feet.	12th dorsal.	yes	yes	drunk	Incomplete.	yes		delirium tremens	yes 1-8% albumen.		in 2 days	yes	*Autopsy.*—Twelfth dorsal vert. was fractured in upper portion, giving rise to an arching backward of spine, and a projection backward of a plate of bone, which in turn had pressed on the cord. This pressure had led to red and white softening for a distance of about 15 mm. Above this was an extravasation between the dura and periosteum.

(Table F.)

Cases treated by Immediate Correction of Deformity, and Fixation by Plaster of Paris Jacket.

Number.	Name. Date. Reference.	Age.	Sex.	Surgeon.	Character of Accident.	Seat of Injury.	Crepitus.	Deformity.	Unconsciousness.	Paralysis.	Pain.	Priapism.	Delirium.	Cystitis.	Bed Sores.	Death.	Autopsy.	Remarks.
66	W. B. April 6, 1884, vol. 119, p. 57.	58	M	Ingalls.	Fell down stairs 20 feet.	Middle dorsal.	yes	yes	no	Complete.	yes		yes	yes	no	11 days.	no	On entrance a plaster of Paris jacket was applied, the patient being suspended by a tripod; deformity reduced by this means; immediate relief from pain; jacket comfortable all the time; could move in bed without pain. Delirium. Death on 11th day, refusing nourishment and stimulants.
14	P. R. July 15, 1884, vol. 121, p. 86.	37	M	Bolles.	Fall.	Neighborhood of last dorsal.	no	yes		Complete.				yes	yes			Splint of plaster of Paris at once applied. Discharged Oct. 17, 1884, relieved. Sept. 23, 1886, letter: Paraplegia; can feel a few inches down from trunk. Legs are crooked and draw up spasmodically. Constipation. Uses catheter. Sits up 4 to 5 hours daily. No medical advice for a year. Now in New Brunswick.
17	F. O. Aug. 24, 1886, vol. 138, p. 181.	21	M	Burrell.	Fall 40 feet.	Lower dorsal 12th.	yes	Gen. curve, with knuckle at 12 d. angle 30°	yes at first.	Complete.	yes							Pat. suspended; deformity reduced as much as possible by pressure; "crunching" of vertebræ felt on reduction; p. of P. jacket applied; sensation in limbs returned immediately. Deformity reduced 11 hours after accident; retention and constipation ceased on 3d day. Sept. 21, patella reflex somewhat exaggerated; no ankle clonus; urine normal. Can move left slightly; r. leg considerably. Oct. 27, '86, has continued to improve in moving legs; can turn in bed; sensation perfect. Nov. 17, '86, p. of P. removed; no patella reflex; no ankle clonus; movement of l. leg good; moves r. toes; cannot lift r. leg; some pain in r. thigh when lifted; strychnia sulph. gr. 1-60, 3td; sits up in steamer chair. Jan. 21, 1887, pat. walking about with aid of chair; power of r. leg slowly improving. Mar. 10, 1887, walks unaided. Apr. 23, '87, walked out in yard; electricity thrice weekly. May 1, '87, examined by Dr. P. C. Knapp; l. thigh and leg, no galvanic reaction; all muscles react to faradic current; r. thigh and leg, no faradic reaction; walks with halt in r. leg. May 4, '87, went home, 129 Chelsea St., Charlestown, Mass. May 5, '87, bar-tender.
81	J. J. Sept. 21, 1886, vol. 139, p. 67.	38	M	Gavin.	Fall 12 feet.	7th cervical.	no	no	no	Complete.	yes	yes	yes	yes	yes	2 mos.	no	Pat. weighed over 200 lbs.; suspended; great pain and dyspnœa; had to be let down; p. of P. applied while suspended; jacket required to be cut up on 2d day, owing to difficulty in breathing; gaping nearly 1 in.; no improvement from jacket; removed on 6th day; abdominal breathing. 3d day, "girdle" sensation. 8th day, paralysis of arms; slough of ear from pressure. 7th day, incontinence of urine and fæces. At end of one month, dribbling of urine. Bed sores, marked emaciation, delirium, death.
18	T. S. Nov. 26, 1886, vol. 140, p. 244.	23	M	Burrell, by courtesy of Dr. M. F. Gavin.	Fall into a sewer, 30 feet.	General curve 7th dorsal to 1st lumbar.	yes	yes	20 minutes.	None.	yes, very severe.							Treated by immediate correction of deformity and fixation by p. of P. jacket. Dec. 5th, plaster splint 2 straps applied. Dec. 8th, jacket removed owing to defective padding; knuckle found at 10th dorsal; jacket reapplied; no paralysis. Dec. 9, discharged, own request, against advice, wearing jacket. Feb. 14, '87, walked into Boston City Hosp. for re-application of jacket: no paralysis or pain; slight deformity at 10 to 12 dorsal vert.

CASE UNDER TREATMENT JUNE 7, 1887.

	C. M. May 17, 1887, vol. 147, p. 76.		M	Gay.	Struck by a falling derrick.	10th dorsal.	yes	angle 45°	yes	Complete.	yes	semi.	no	yes	yes	under treatment		Within a few hours of the accident; pat. was etherized; suspended; deformity was partially reduced, and a p. of P. jacket with shoulder straps was quickly applied. Jacket was uncomfortably tight; 3d day split up over abdomen; 4th day completely, owing to tympanites. Moved toes 2d day, slightly right foot. Has had and has now hyperæsthesia of the legs; this is diminishing. Moved right leg voluntarily the 13th day; left leg as bad as ever. Bed sore over sacrum, also over calf of r. leg. Turns on either side; the jacket very comfortable; cystitis better.

missible, for the thoracic viscera, aorta, solar plexus, and vena cava would forbid; and an attempted removal from the side would be equally inexpedient.

Five cases have been operated upon at the Boston City Hospital, and they appear in Table D of Operations. All ended fatally.

It may be of interest to see the status that this procedure should occupy in Surgery. Paré, Heister, and many of the older authors discussed the propriety of excising portions of the vertebræ or trephining, but Henry Cline[8] first performed the operation at St. Thomas's Hospital, June 16th, 1814. The patient lived seventeen days, and Mr. Cline admitted that the operation hastened the end. Lidell[9] failed to find a single well authenticated successful case. It is certainly true, that Legoust,[10] Jobert,[11] E. Gurlt,[12] Hamilton[13] and Sir Charles Bell[14] all condemn the operation in unqualified terms.

Gun-shot injuries, however, may be excepted from this sweeping condemnation, and all the reported successful cases of operation, closely resemble that done by Louis[15] in 1762, where bony fragments were removed after a gun-shot fracture of the spine.

The removal of fragments after gun-shot injuries to the spine is perfectly justifiable, and will, I believe, give a fair measure of success. In the War of the Rebellion[16] "there were twenty-four cases of removal of fragments of the vertebræ after gun-shot fracture, with fatal results in only

[8] New England Journal of Medicine and Surgery, Vol. IV., No. 1, January, 1815.

[9] On injuries to the Spine, American Journal Medical Sciences, October, 1864; Vol. XLVIII. p. 320.

[10] Chirurgie d'Armee, pp. 341, 352.

[11] Plaies d'Armee a feu, Paris, 1833, p. 125.

[12] Handbuch der Lehre von dem Knochenbrüchen Hamm. 1864, p. 186.

[13] Treatise on Fractures and Dislocations, p. 187.

[14] Observations on Injuries of the Spine and of the Thigh Bone, 1824.

[15] Rèmarques et Observations sur les Fractures et la Luxation des Vertebræ, Mem. Path. Arch. Gen. de Med., 1836, LXI. 2 Série, p. 417.

[16] Hist. of the War of the Rebellion, Pt. 1, Surg. Vol., p. 459.

ten instances." In nine instances, however, of the fourteen examples of recovery, the spinous process, or fragments of it, only were removed. In the five cases of recovery, in which portions of the laminæ or the transverse processes were removed, the results were much less satisfactory, nearly all of the patients having serious disability.

On the other hand, the operating on fractures of the spine, not compound, is not a justifiable measure, and without further evidence supporting this operation, it will have to be placed among the impracticable efforts of experimental or venturesome surgery.

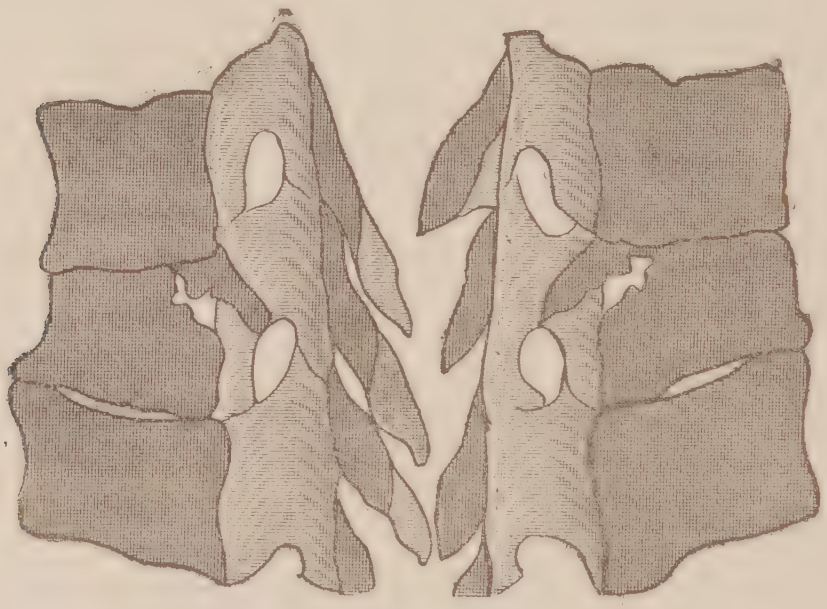

PLATE III.

c. IMMEDIATE RECTIFICATION OF THE DEFORMITY IN FRACTURE OF THE SPINE AND FIXATION BY PLASTER OF PARIS JACKET.—On the 10th of August, 1886, I saw the autopsy of a woman who had been under my care, with a fracture of the spine, in the Boston City Hospital (Plate III. Case 78), and found that a plate of bone from the posterior wall of the body of the 12th dorsal vertebra had been broken off, in addition to an arching backward of the whole spine. This plate had pressed upon the cord, and had, in 38 hours,

led to red and white softening for a distance of 15 mm. The presence of the plate *anterior* to the cord showed me that an operative procedure would have been of no avail, and the *softening* occurring so early in the cord, led me to believe that, if aught was to be done to remedy the damage caused by a fracture of the spine, it must be at once.[17]

The arching of the vertebræ suggested that the difficulty might be overcome by *immediately* pressing back the deformity, and fixing it in this corrected position.

I determined to act upon this principle in the next case. On Aug. 24th, 1886, Case 17 was admitted, and was seen within twelve hours of the time of his fall of 40 feet. The risks of immediate rectification and suspension having been explained to him, he was suspended, as represented in Plate IV., with this difference, that the tripod was placed over the head of the bed, a ward-master was placed upon a small table at either side of the patient to lift up on the body at the axillæ. This, when one has many assistants, is a great aid to the patient. The back of the patient, while being changed from the horizontal to the perpendicular position, should be carefully supported, and when the patient is brought into an erect position the buttocks are free from the table.

The deformity, which was at an angle of at least 30° and included the 12th dorsal vertebra, was reduced, and a plaster of Paris jacket was quickly applied. An anæsthetic was not given, for I did not wish to have any danger masked. The patient's sufferings during the suspension, rectification and application of the jacket, were more intense than anything I have ever seen; he nearly collapsed, but the jacket was finished, and stimulants were given. On recovering himself, he said that there had been an immediate return of sensation in his limbs, directly following the reduction of

[17] Case 80. Red softening in cord. Death in 18 hours. Table of Autopsies, E.

the deformity. His recovery proceeded uninterruptedly. On April 23d, 1887, for the first time he walked out. Dr. P. C. Knapp examined him on May 1st, 1887, and found

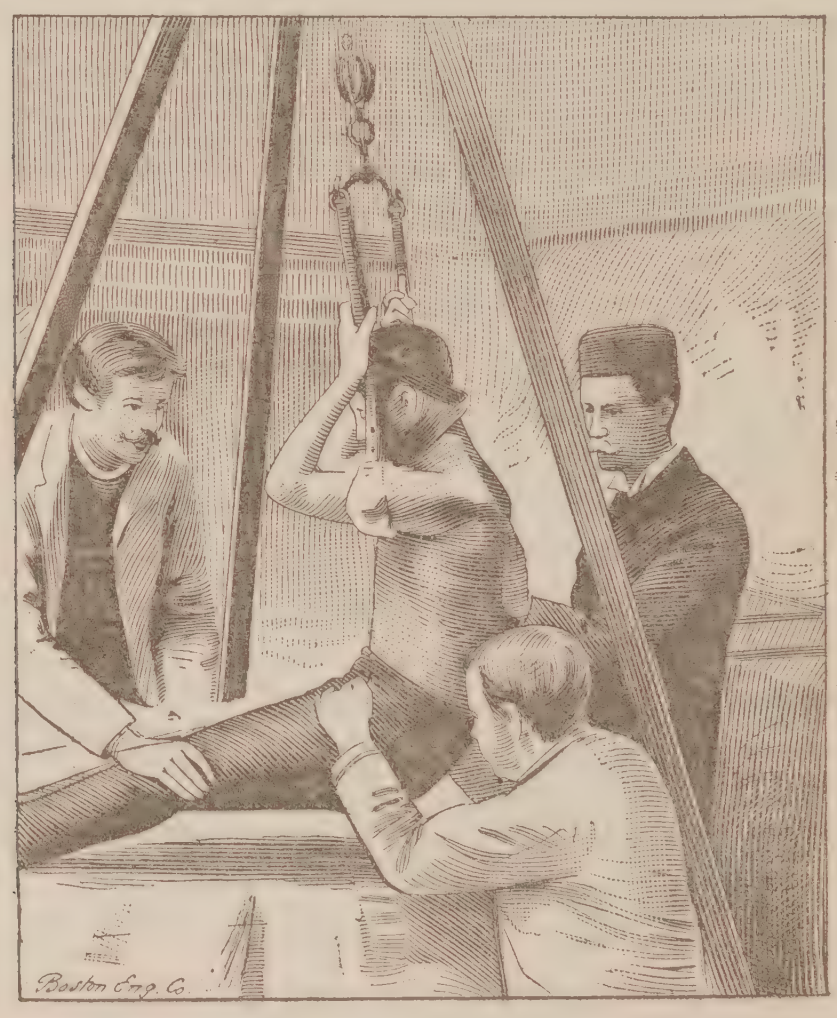

PLATE IV.

in the left thigh and leg no galvanic reaction. All muscles react to faradic current. The right thigh and leg have no faradic reaction. He now walks with a halt in the right leg, and on May 5th, 1887, was found acting as a bar tender. Plates V. and VI. represent his present condition.

This procedure at the time I supposed to be an original thought, but soon found that other gentlemen in the same hospital had preceded me.

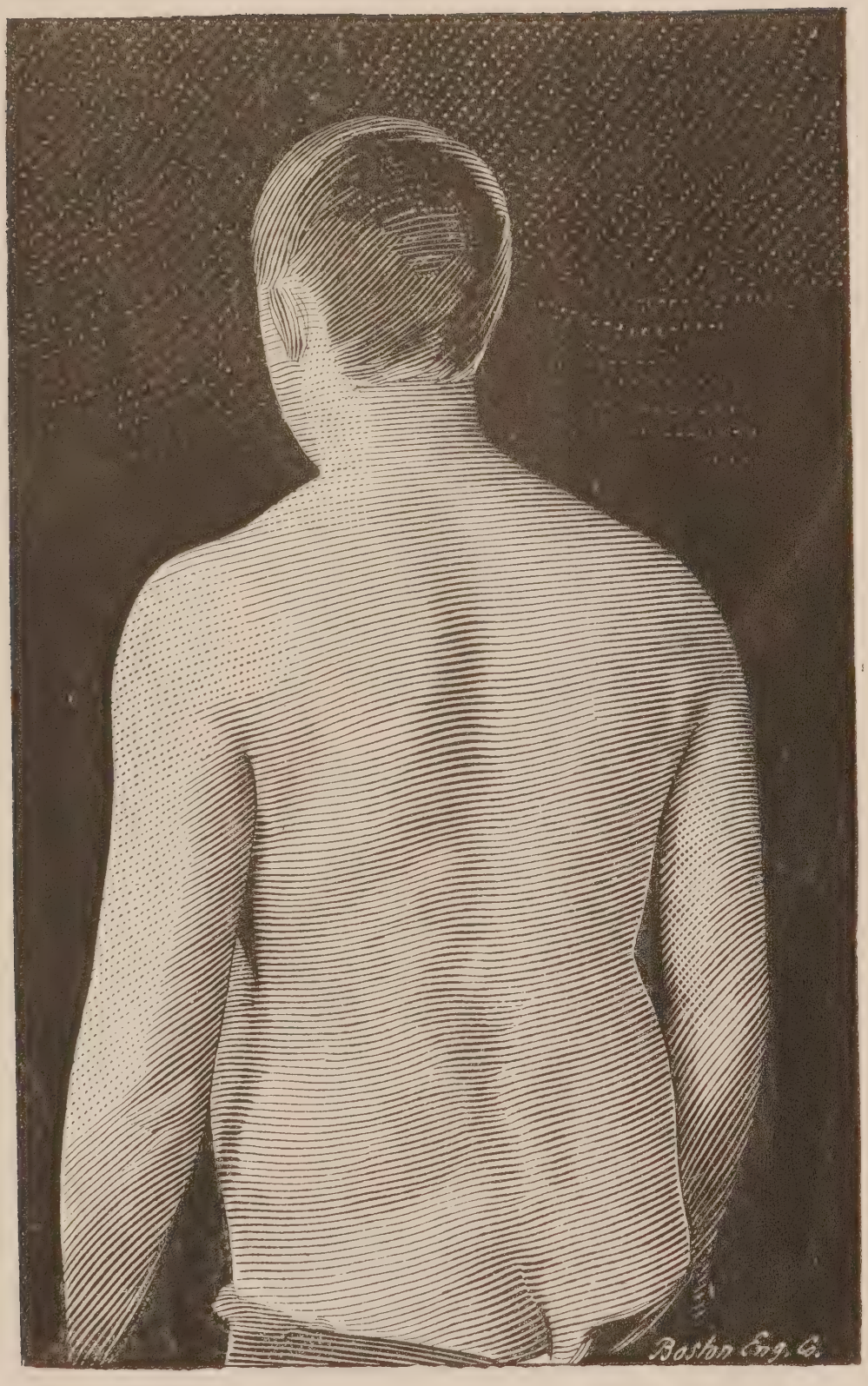

PLATE V.

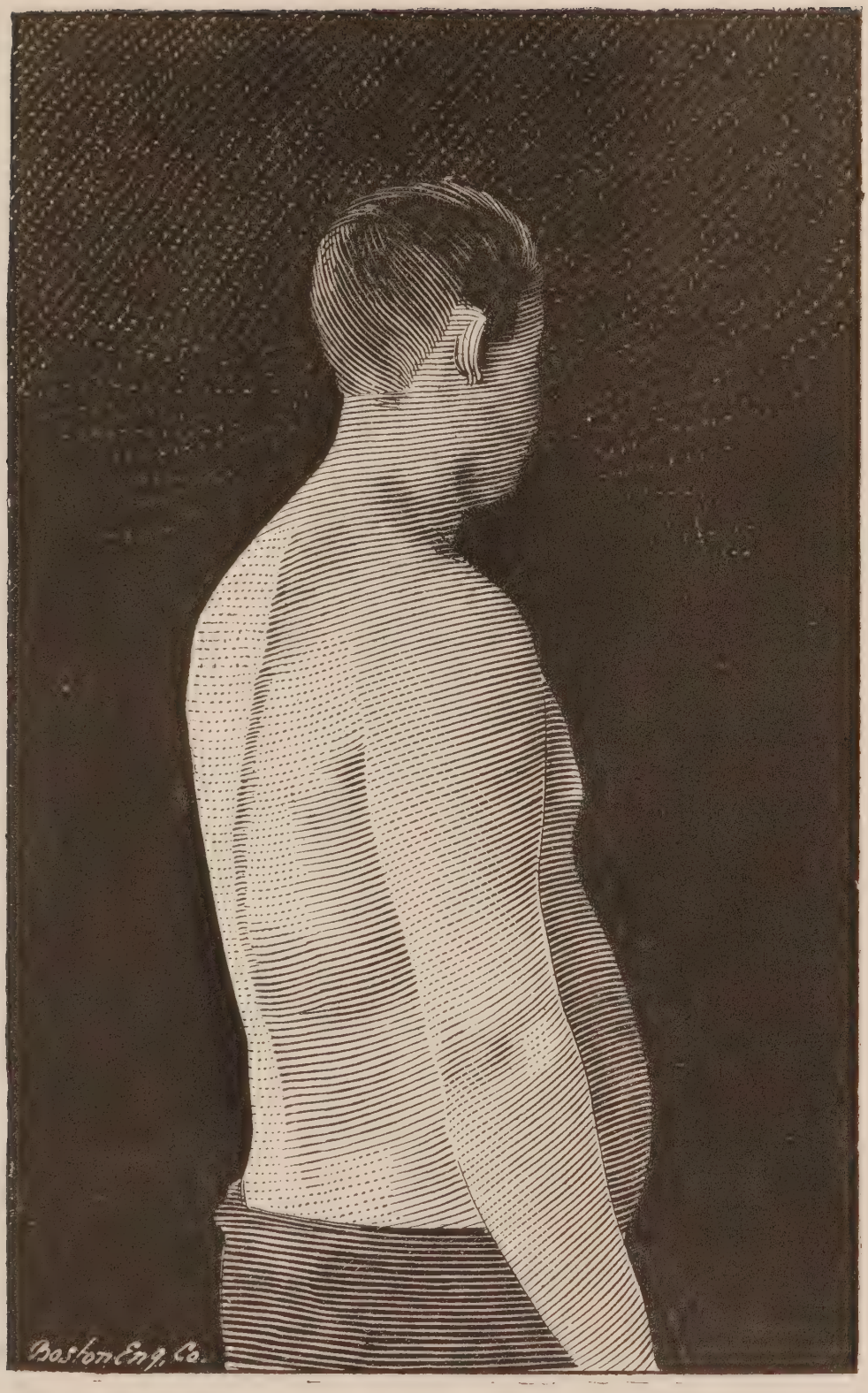

PLATE VI.

Five cases has been treated in this manner in the hospital to a completion. Dr. Wm. Ingalls (Case 66, Table F) on April 6th, 1884, applied a jacket to a fractured spine, the deformity having rectified itself by suspension, but his patient unfortunately died. Dr. W. P. Bolles (Case 14, Table F) on July 15th, 1884, applied a splint of plaster of Paris at once to a fractured spine, and his patient recovered, but is useless. Dr. M. F. Gavin (Case 81, Table F) on Sept. 21st, 1886, rectified the deformity in a fractured spine, having suspended a patient weighing over 200 lbs. Great dyspnœa and pain occurred. The jacket did absolutely no good, requiring to be cut up on the second day, and gaped nearly an inch. The patient died in two months, of exhaustion.

On Nov. 26th, 1886 (Case 18, Table F), by the courtesy of Dr. Gavin I was again enabled to attempt the rectification of a fracture of the 10th dorsal vertebra, and applied a plaster of Paris jacket; this case was seen a short time after the fracture occurred. There was no paralysis, and the patient made a good recovery.

Of these five cases two have died, one is useless, and two have recovered. (See Table F.)

Of the expediency of rectification of the deformity, I can but think that an important lesson can be learned from a study of these drawings of specimens from the Warren Anat. Museum. Plates I. and II. show that any efforts would be futile; while Plates IX., X., XI., XII. and XIII. are very suggestive as to the possibility of immediately rectifying the deformity.

The application of the plaster of Paris jackets for fracture of the spine is by no means a new idea, and the literature on the subject at my command gives me the following facts:

In the fall of 1874 Professor Sayre states that he first applied a plaster of Paris jacket for Pott's disease; and

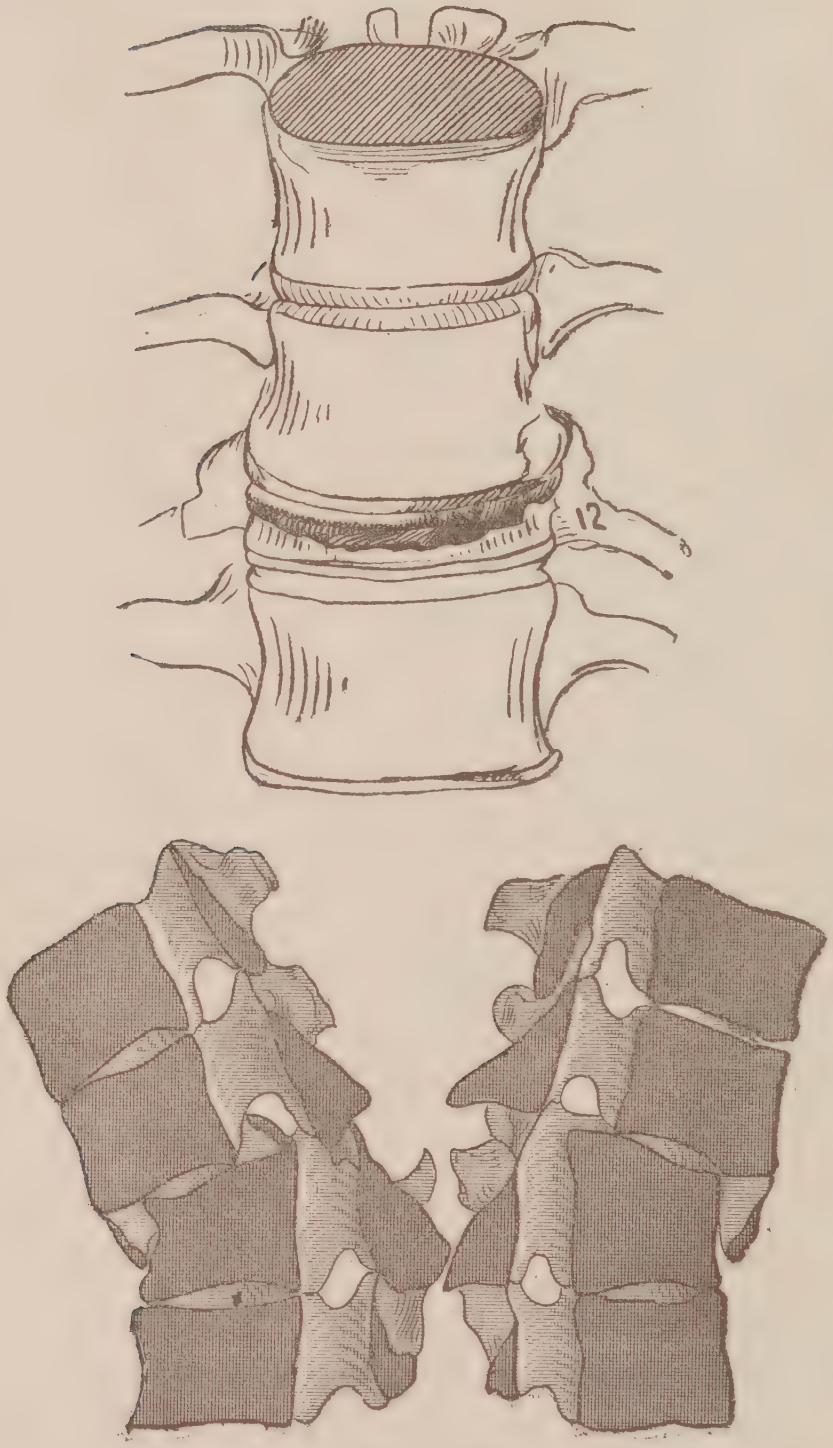

Plate IX.

(No. 140 Warren Museum,)

Fracture of 12th dorsal, with displacement; extensive laceration of inter-vertebral substance; cord almost completely severed; death, 9½ weeks; paralysis, bed sores, cystitis.

(Table G.) TABLE OF DEATHS.

Number.	Name. Date. Reference.	Age.	Sex.	Surgeon.	Character. of Accident.	Seat of Injury.	Crepitus.	Deformity.	Unconsciousness.	Paralysis.	Pain.	Delirium.	Cystitis.	Bed Sores.	Deaths.	Autopsy.	Priapism.	REMARKS.
19	J. S. March 21, 1866, vol. 7, p. 8.	28	M	Buckingham.	Fall.	Upper lumbar.		yes		Complete.	yes	no	yes	yes	Yes Jan. 10, 1867.		no	Treatment—rest in bed; electricity; strychnia, sulph. gr. 1-60 3id. Discharged well, Dec, 4, 1866. Letter from brother May 15, 1887. states that patient died Jan. 10, 1867, from exhaustion, bed sores and inflammation of bladder.
20	M. B. June 15, 1866, vol. 10, p. 57.	21	M	Cheever.		Cervical.	yes	yes	yes	Complete.	yes	no	no	no	June 16, 1866.	no	no	
21																		(See Table of Operations.)
22																		" " " "
23																		(See Table of Operations and Autopsies.)
24	C. C. Oct. 16, 1868, vol. 19, p. 220.	37	M	Thaxter.	Blow on head from falling bale of cotton.	Dislocation of atlas. forward and fracture.	no	yes	no	Complete.	yes				4th day.	no	no	Respiration abdominal; all efforts at reduction futile; death from suffocation.
25	T. C. July 20, 1869, vol. 26, p. 14.	52	M	Ropes.	Pack of leather (150 lbs.) fell on back while stooping.	Cervical.				Complete.	yes				36 hours.	no	no	
26	W. N. Jan. 30, 1870, vol. 25, p. 241.	33	M	Cheever.	Fell 3 stories, striking on back.	Cervical.	no	yes	no	Complete.	yes				Next day.	no	yes	
27	J. M. April 19, 1870, vol. 28, p. 114.	24	M	Thorndike.	Fell from scaffold.	Injury to head, fracture 8th dorsal.	yes	yes	no	Complete.	yes	no	no	no	4 days.	no	no	Much deformity at 8th dorsal; abdominal breathing.
28																		(See Table of Autopsies.)
29	J. D. Nov. 6, 1870, vol. 33, p. 1.	23	M	Thaxter.	Fell 50 feet 9 days before entrance.	Lower lumbar.	no	no	yes	Partial.	yes		yes	yes	18th day after injury.	no		
30	M. F. Jan. 11, 1872, vol. 46, p. 216.	27	M	Thorndike.	Fell 10 feet striking on back.	9th, 10th and 11th dorsal.	yes	yes	no	Complete.	yes	yes	yes	yes	25 days.	no	yes	Day after entrance etherized; deformity reduced by extension and counter-extension, and pressure over deformity; trunk secured to head of bed, and 15 lbs. of extension applied to legs. In 1 week, delirium tremens: incontinence of urine. Failed gradually; death from exhaustion.
31	C. S. July 5, 1872, vol. 41, p. 269.	33	M	Ingalls.	Fell while intoxicated from 3d story window.	Multiple injuries; upper dorsal.		yes	no	Complete.	yes				In 1 week.	no	yes	
32	J. H. Aug. 3. 1872, vol. 43, p. 65.	50	M	Ingalls.	Fell 9 days before entrance.	11th and 12th dorsal.	yes	yes	no	Complete.	yes		yes	yes	1 month.	no	no	Exhaustion.
33																		(See Table of Autopsies.)
34	P. S. Oct. 21, 1872, vol. 43, p. 204.		M	Gay.	Intoxicated; wheel over neck.	6th and 7th cervical.	no	no	no	None at first.	yes				2d day.	no	yes	On entrance, complained of pain in neck; physical exam. negative. In 3 hours legs became paralyzed; abdominal respiration; later, complete paralysis; great thirst; death. Distinct crepitus felt after death, between 6 & 7 cerv. vert.
35																		(See Table of Autopsies,)
36	O. B. July 16, 1873, vol. 47, p. 154.	40	M	Ingalls.		4th and 5th cervical.	yes	yes	no	Below 3d ribs.	yes				Next day.	no	yes	
37	M. F. Oct. 20, 1873, vol. 52, p. 14.	38	F		Fell from 2d story, admitted following day.	Dorsal.			yes	Complete.			yes	yes	3 months.	no		

TABLE G (Continued).

38	M. M. June 12, 1874, vol. 56, p. 26.	38	M	Homans.		Cervical.	yes	yes	no	Below 2d ribs.	yes	no	no	no	24 hours.	no	yes	
39	J. T. Aug. 13, 1874, vol. 53, p. 113.	50	M	Gay.	Tossed by a bull.	3d or 4th cervical.	yes	yes	no	Complete.	yes				Same day.	no		Respiration abdominal; thirst. Died suddenly, speaking 5 minutes before death.
40	E. H. Sept. 2, 1874. vol. 53, p. 143.		M	Gay.	Fell 30 feet.	Lumbar, fracture of pelvis.	yes	yes		Absent.	yes				Next day.	no		
41	E. T. May 3. 1875. vol. 57, p. 270.	33	M	Ingalls.	Fell 18 feet.	7th or 8th dorsal.	yes	yes	no	Complete.	yes		yes	yes	52 days.	no		Incontinence of urine and fæces.
42	J. W. Sept. 12, 1875, vol. 39, p. 271.	34	M	Gay.	Fell 8 feet.	Mid. dorsal.	yes	yes		Complete.	yes		yes	yes	In 1 month.	no	no	Exhaustion.
43																		(See Table of Autopsies.)
44	W. F. Nov. 10, 1875, vol. 64, p. 84.	15	M	Thorndike.	Wheel of cart passed over chest.	4th and 5th dorsal.	yes	yes	yes	Complete.	yes				18 hours.	no		Fract. 3 ribs on right side.
45	J. M. Sept. 26, 1873, vol. 52, p. 14.		M	Fifield.	Fell 2 stories striking on back.	2d and 3d lumbar.	yes	yes	yes	Complete.	no				4 hours.	no		
46	B. M. Jan. 1, 1877, vol. 68, p. 227.		M	Thorndike.	Struck by a falling basket of sand.	Multiple injuries 4th lumbar.		yes							½ hour after admission.			
47	J. F. Oct. 30 1877, vol. 74, p. 132.	45	M	Fifield.	Struck on head by chain of coal scuttle; fell some distance.	Lumbar; Multiple injuries.		yes	no	Complete.	yes				4th day.	no	yes	Incontinence; spinous processes of lower lumbar vert. deflected to one side, and a space existed between 4 and 5 lumbar large enough to lay finger into tympanites; no dejection, even from ol. tiglii gtt. iv.
48	J. K. Aug. 25, 1878, vol. 42, p. 126.	32	M	Thorndike.		12th dorsal.	yes	yes	no	Complete.	yes				Following day.	no	yes	
49	J. M. June 30, 1879, vol. 84, p. 77.	23	M	Homans.	Fell into a vessel's hold.	7th dorsal.	yes	yes	no	Complete.	yes	no	yes	yes	9 days.	no	yes	Tympanites; abdomen tapped with relief.
50	M. P. Aug. 13, 1879, vol. 83, p. 170.	40	M	Gay.	Fell down 10 steps.		yes in lower cerv.			Complete.	yes	no	no	no	Next day.	no	yes	Tympanites relieved by rectal tube.
51																		(See Table of Autopsies.)
52	M. D. Oct. 10, 1879, vol. 86, p. 58.	45	F	Fifield.	Fell 3 stories.	Lumbar.	yes	yes	no	Complete.	yes	no	no	no	6 days.	no		Tympanites relieved by rectal tube.
53	J. H. May 22. 1880. vol. 87, p. 252.	51	M	Ingalls.	Fell 35 feet.	Lumbar.		yes		Complete.					Next day.	no		Death sudden.
54																		(See Table of Autopsies.)
55	P. B. Oct. 14, 1880, vol. 91, p. 21.	42	M	Gay.	Fell 15 feet.	Upper lumbar.	yes	yes	no	Complete.	yes	no	no	no	3d day.	no	no	Death said to be due to œdema of the lungs.
56	P. W. G. Feb. 10, 1881, vol. 91, p. 120.	41	M	Thorndike.	Elevator struck patient on back, "doubled him up."	12th dorsal.	yes	yes	no	Complete.	yes	yes	yes	yes	79 days.	no		Etherized, and by extension, counter-extension and pressure over deformity, the deformity was nearly reduced; kept in position by pads; 10 lbs. extension and tight canvas belt; incontinence; 40 days after entrance, plaster of P. jacket applied; pat. grew steadily worse; death from exhaustion.
57																		(See Table of Autopsies.)
58	J. S. Aug. 22, 1881, vol. 98, p. 109.	32	M	Fifield.	Fell 30 feet.	Multiple injuries, 2d lumbar.	yes	yes	no	Complete.	yes				5 days.	no		Cause of death obscure.

TABLE G (Concluded).

59																		(See Table of Autopsies.)
60	E. W. May 6, 1882, vol. 104, p. 91.	38	M	Homans.	Fell 65 feet.	Multiple injuries, middle dorsal.	no	no		Complete.	yes		yes		8 days.	no		Died suddenly, without cause being known.
61	D. C. May 10, 1882, vol. 104, p. 106.	39	M	Homans	Fell 30 feet striking back.	1st lumbar.		yes		Complete.	yes	yes	yes	yes	21 days.	no		Œdema of penis; girdle sensation; tympanites.
62																		(See Table of Autopsies.)
63																		" " " "
64	J. S. Aug. 8, 1882, vol. 106, p. 246.	39	M	Thorndike.	Struck on back by a boom.	8th dorsal.	no	yes	semi	Complete.	yes		yes		8 days.	no		Severe pain, with girdle sensation; vomiting.
65	J. C. Aug. 10, 1883, vol. 114, p. 219.	17	M	Thorndike.	Fell 40 feet.	Multiple injuries, double fracture mid. dorsal.	yes	yes	yes	Complete.	yes		yes	yes	4 months.	no		No special treatment; death fram exhaustion.
66																		(See Table for Immediate Rectification of Deformity and Fixation by P. of P. Jacket.)
67	J. C. May 22, 1884, vol. 122, p. 8.	30	M	Homans.	Struck by a boom.	4th and 5th dorsal.	yes	yes	yes	Complete.					24 hours.	no		Concussion of brain; respiration diaphragmatic.
68	F. D. June 12, 1884, vol. 119, p. 269.	25	M	Post.	Railroad accident	Multiple injuries, fract. of mid. dorsal.	yes	yes	yes	Complete.	yes				Few hours.	no		Pulseless at time of admission.
69	W. E. Aug. 2, 1884, vol. 121, p. 132.	65	M	Gay.	Fell down stairs.	6th cervical.	no	yes	no	Complete.	yes		yes	yes	6 days.	no	yes	Cyanosis; diaphragmatic respiration.
70																		(See Table of Operations.)
71																		" " " "
72	J. C. Jan. 5, 1885, vol. 126, p. 92.	65	M	Gay.	Fell from wagon, struck on face.	Cervical.		yes		Complete.	yes	yes	yes	yes	11 days.	no	no	
73																		(See Table of Autopsies.)
74	C. S. Sept. 15, 1885, vol. 130, p. 234.	25	M	Bolles.	Fell from 2d story window.	Dorsal.		yes		Complete.	yes				Same day.	no		
75	E. C. Jan. 6, 1886, vol. 132, p. 261.	42	M	Bolles.	Carriage fell on patient.	4th cervical.	yes	yes	yes	Complete.					Next day.	no		Asphyxia.
76	E. C. Dec. 3, 1886, vol. 142, p. 12.	45	F	Bradford.	Fell 2 stories.	Cervical.	yes	no	at first	Absent.					15 hours.			Spoke five minutes before death.
77	M. C. July 31, 1886, vol. 138, p. 108.	21	M	Bolles.	Railroad injury, multiple.	6th dorsal.	yes	yes	no	Complete.	yes				3 days.	no		No re-action from shock.
78																		(See Table of Autopsies.)
79	B. F. E. Oct. 26, 1886, Carney Hosp.	50	M	Burrell.	Fall from coal bridge 18 feet, striking on back.	9th and 10th dorsal.	yes	yes	no	Complete.	yes	yes	yes	yes	212 days.	no	no	Was transported from Lake Superior to Boston on an air bed: cystitis so severe that it perforated the recto-vesical septum, causing recto-vesical fistula. Complication of dribbling of urine from anus, relieved by glass rectal urinal.
80																		(See Table of Autopsies.)
81																		(See Table for Immediate Rectification of Deformity and Fixation by P. of P. Jacket.)
82	A. M. April 11, 1887, vol. 145, p. 176.	44	M	Gay.	Fell 15 feet.	Lower cervical and upper dorsal.	no	no	no	Complete.	yes	no			40 hours.	no	yes	Fracture bed; tympanites; diaphragmatic respiration. This may be a case of hemorrhage into or about the cord.

doubtless to his bringing this method of treatment of Pott's disease[18] before the profession is due the present application of plaster of Paris jackets in fresh fractures of the spine.

On June 25th, 1879, J. R. Weist, of Richmond, Indiana (see Appendix I.), reduced a fracture of the 9th

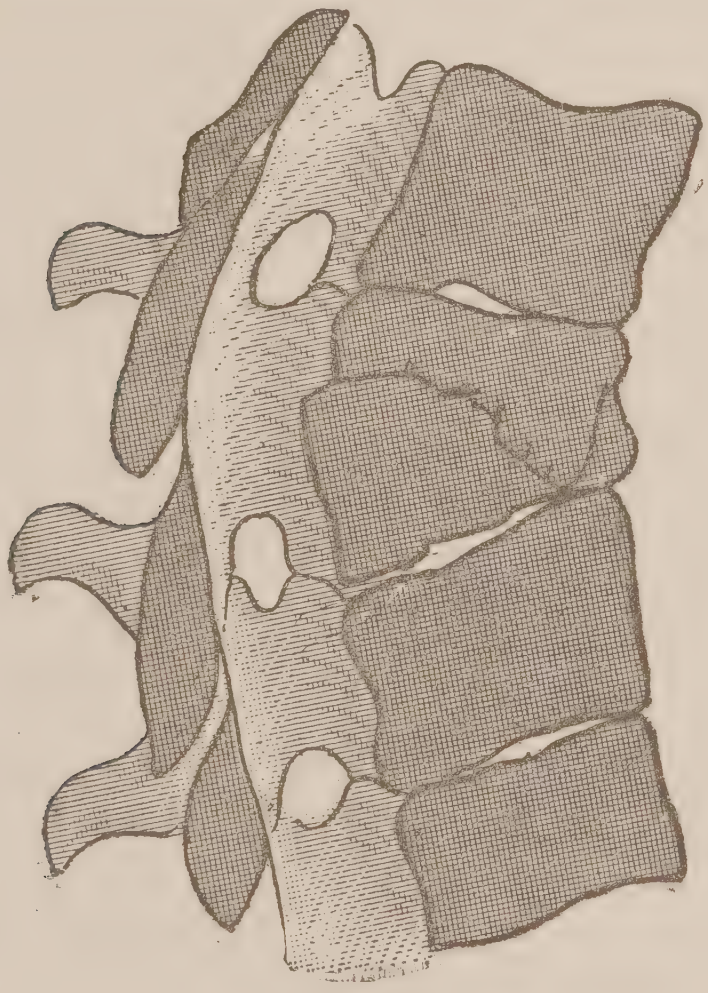

PLATE X.

(No. 1376 WARREN MUSEUM.)

Diagram of a fracture of the body of a vertebra, not narrowing the canal materially.

dorsal vertebra during suspension, and applied a plaster of Paris jacket. This was followed by a great relief to the pain, uninterrupted improvement, and a recovery of the patient on the 67th day.

[18] Succinct History of the Plan of Treatment of Pott's Disease by Suspension and the use of Plaster of Paris Bandage, p. 4.

Dr. Weist says that when he applied the plaster dressing in this case, he was not aware that it had been used in such cases; but he learned from Dr. Sayre that it had been

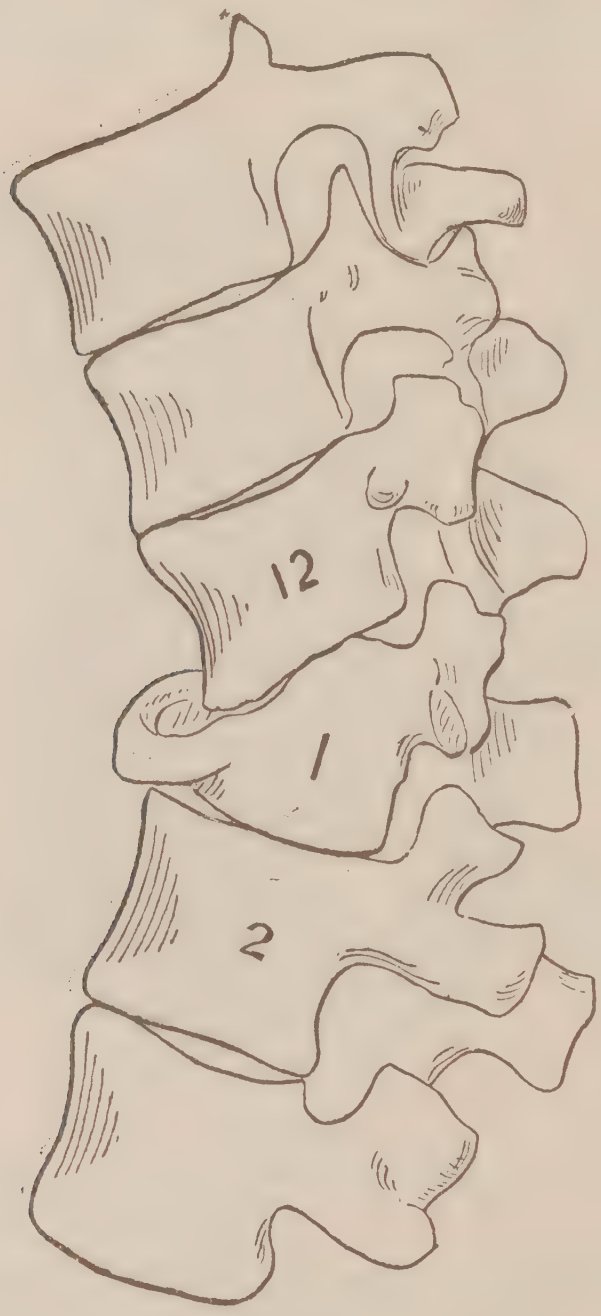

PLATE XI.

(No. 941 WARREN MUSEUM.)

Fracture of 1st lumbar vert. Female, æt. 19; fall, 20 ft., striking on nates. Spinal canal encroached upon. Symptoms.—Complete paralysis, immediate. Results,—Moderate improvement after 3 weeks; power of sphincters regained.

used several times in a similar manner at Bellevue Hospital with very satisfactory results, and says that probably others also have used the plaster jacket in this way. However this may be, Dr. Weist made the report at the

PLATE XII.

(No. 4629 WARREN MUSEUM.)

Old fracture 12th dorsal. Upper edge has been broken away from body of centrum. 12th dorsal vertebra is crushed anteriorly. Considerable narrowing of canal, opposite upper edge of 12th dorsal vertebra.

C. A., æt. 18; fell on plank floor. Paralysis, bed sores, cystitis; death after 2 8-12 years. Cord considerably disorganized at seat of injury.

request of Professor Sayre, and with the exception of an allusion to a case of Spratley's by Reginald Harrison, shown at the Liverpool Medical Institution,[19] Dr. Weist's case is the earliest published record of the procedure that I have been able to find.

[19] Surgical Diseases of the Urinary Organs (Lectures on), p. 51, by R. Harrison, at the season 1878-9.

In the remainder of the year 1879, I find five cases treated in this manner. König, of Gottingen, in 1880 said that having used suspension and plaster of Paris jackets for caries of the vertebræ, where there was paralysis,

PLATE XIII.

(No. 938 WARREN MUSEUM.)

Fracture 4th dorsal; longitudinal section, showing backward displacement and narrowing of canal. Female, æt. 25; fall, 15 feet; paralysis (complete); bed sores; death, 9 weeks.

with great improvement, he was led to apply a similar treatment to fresh fractures of the spine. He did not advocate its employment in all cases, but in his three cases the results were favorable. (See Appendix II.)

He said that doubtless the method had been employed in other hospitals, and he wrote the paper as a contribution to the subject. Wagner, of Königshutte, reported two cases with which he was not at all satisfied. In both cases he was obliged to remove the jacket. (See Appendix III.) He advised caution in the use of the jacket, had seen it produce paralysis, and felt that the replacement might produce alarming or dangerous symptoms. He advocated its application at the expiration of fourteen days. The whole subject was discussed at the German Congress of Surgeons in 1881,[20] and was evidently considered a measure worthy of trial in picked cases. Langenbeck mentioned a case which he had treated in this manner in 1862, and which ended in failure. Since then I have accounts of the following cases: one by Berkeley Hill (see Appendix IV.); one by Edouard de Reynier (see Appendix V.); one by Carson (see Appendix VI.); two cases by H. O. Marcy, private letter (see Appendix VII.); and the five cases included in Table F.

No attempt has been made to investigate cases where a jacket has been applied after the tenth day following an injury. This gives us sixteen cases, in which three died, three derived no benefit from the method, and ten were greatly benefited.

The subject may be summarized, and I submit the following conclusions:

First—That, in the *immediate* correction of the deformity and fixation with plaster of Paris jacket or other means, we have a rational method of treating a large number of cases of fractures of the spine.

[20] Berl. Klin. Wochen., 1881, p. 247.

Second—That, considering the hopelessness of results in fracture of the spine when treated expectantly, almost any risk is justifiable.

Third—That the *immediate* correction of the deformity is imperative, *if* softening of the cord can and does occur from pressure at the end of forty-eight hours.

Fourth—That the suspension of the patient is only a means of rectifying the deformity; that certain fractures could be simply pressed into position while the patient lies prone or supine.

The objections to the treatment are,—

1st. That the expectant plan of treatment gives a small percentage of recoveries.

2d. That there are serious risks, especially in the cervical region, attending the suspension of a patient and the rectification of the deformity with a fractured spine, in the way of shock, collapse and death.

3d. That in attempting to relieve pressure on the cord, by rectifying the deformity, we might either sever the spinal cord or make pressure upon it. This is a matter of chance.

My own belief regarding the status which the procedure should occupy in Surgery is, that it will occasionally be a life saving measure; that it should be applied under anæsthesia in all cases of fracture of the spine, which are not conclusively known to be irremediable; and that apart from the chance of recovery offered to the patient by this means, it will almost invariably make the patient more comfortable, in that he can be handled more easily.

In conclusion I wish to acknowledge my indebtedness to Drs. H. W. Cushing, E. G. Brackett and W. H. Prescott for valuable assistance rendered in preparing details of this paper.

(Table H.) TABLE OF RECOVERIES.

Number.	Name. Date. Reference.	Age.	Sex.	Surgeon.	Character of Accident.	Seat of Injury.	Crepitus.	Deformity.	Unconsciousness.	Paralysis.	Pain.	Priapism.	Delirium.	Cystitis.	Bed Sores.	Death.	Autopsy.	REMARKS.
1	N. K. Jan. 30, 1866, vol. 8, p. 152.	14	F	Coolidge.	Fell 3 weeks before entrance.	Lumbar.		yes										Treatment—rest in bed. Discharged relieved, Feb. 16, 1866.
2	J. R. June 20, 1867, vol. 11, p. 136.	30	M	Cheever.	Fell from mast head.	Fracture of spinous process in lumbar region.		yes		Retention, Constipation.	yes							Discharged well in one month.
3	W. K. Oct. 29, 1870, vol. 32, p. 24.	19	M	Fifield.	Fall from ladder.	1st dorsal.	yes	no	no	Yes.	yes	no	no	no	no	no	no	Discharged Dec. 24, 1870, nearly well; nothing especial done.
4	H. W. Oct. 2, 1874, vol. 53, p. 211.	50	M		Fall 25 feet.	Lower dorsal.	yes	yes	no	Complete.	yes		yes	yes	yes			Some nausea and vomiting; tympanites; paracentesis abdominis; involuntary dejections; bed sores on left flank and thighs, exposing muscles; catheter kept in bladder. Heavy drinker. Discharged relieved, Dec. 30, 1874. No record of paralysis; bed sores clean and small.
5	D. F. Dec. 3, 1874, vol. 55, p. 102.	55	M	Gay.	Buried in débris by giving way of a floor.	Lower dorsal.	yes	yes	no	Complete.	yes			yes	yes			Tympanites; incontinence of urine; diarrhœa; became fretful; circulation poor; no improvement in paralysis. Discharged relieved.
6	J. E. Feb. 26, 1878, vol. 76, p. 130	38	M	Thorndike.	Fell down 4 steps, striking on head and shoulders.	Cervical.	yes	yes	no	Partial, at first.	yes							Paralysis of arm, hand and leg at once, lasting 12 hours; gradual return of sensation; probable fracture of spinous process. Discharged March 23, 1878, relieved.
7	P. W. June 21, 1881, vol. 95, p. 136.	32	M	Ingalls.	5 weeks before entrance fell quite a distance.	Lumbar.		yes		Complete.	yes			yes	yes			Discharged in one month, condition unchanged; incontinence of fæces and urine.
8	F. D. Sept. 2, 1882, vol. 106, p. 221.	34	M	Thorndike.	Fell 2 stories.	Lumbar, 4th.	yes	yes	no	Complete.	yes		no	yes	yes			Tympanites; involuntary dejections; reflexes increased; considerable twitching; "girdle" sensation; urethritis. Treatment, fracture bed. Oct. 14, 1882, motion in legs. Nov. 9th, sat up. Nov. 13, discharged, condition relieved. Seen on Oct. 1, 1886. Staid in house 3 yrs. 8 mos., going about on crutches; retention for 5 mos.; incontinence of fæces 2 years. Present condition—slight incontinence; can go down stairs fairly well with crutches; can move r. leg fairly, but subjectively heavy; almost complete motor paralysis of l. leg; appetite fair; was very fat for a time, now weight nearly normal.
9	T. K. Sept. 13, 882, vol. 108, p. 18.	32	M	Thorndike.	Fell from team, striking on face.	Cervical.	yes	no	no	Absent.	yes							Mounted team and rode home; neck became stiff. Examination—head carried low; crepitus on lateral pressure of transverse processes of 5th & 6th cerv. vert. Sept. 14, 1882, p. of P. jacket, with a hood about head; back fixed with ham splint, attached with shoulder cap over head, after partial removal of plaster. Sept. 29, 1882, "bound to go"; plaster removed; projection felt over 5 & 6 cerv. spines; some tenderness: moves head easily. No further symptoms.
10	R. B. Oct. 5, 1882, vol. 108, p. 87.	29	M	Fifield.	Was caught between the seat of a team and the top of a driveway.	Lumbar, 1st & 2d.	no	yes	no	Absent at first, slight numbness later.	yes							Delirium tremens on 4th day. Discharged Nov. 1, 1882, nearly well; projection plainly felt.
11	S. C. Oct. 5, 1883. vol. 116, p. 92.	33	M	Fifield.	Fell 14 feet, striking head.	Spine of 6th cervical.	yes	no	no	Absent.	yes							Discharged well in 6 weeks; plaster hood applied 3d day. Letter, May 16, 1887, does light work on a farm.
12	M. C. Nov. 23, 1883, vol. 115, p. 165.	40	M	Cheever.	Thrown from a team.	Upper Lumbar.	yes	yes	no	Complete.	yes	no		yes	yes			Dec. 22, 1883, thinks he can do as well at home as in hospital; discharged, own request, unrelieved.
13	W. R. July 7, 1884, vol. 122, p. 159.	45	M	Homans.	Thrown from railroad bridge.	Dorsal, 4th & 5th.	yes	yes	For a few minutes.	Absent.	yes							Lobar pneumonia 36 days after entrance. Treatment—fracture bed, swathe round body. Discharged, well, Aug. 30, 1884. May 18, 1887, walks about, and does a little work.
14																		(See Table for Immediate Rectification of Deformity and Fixation by P. of P. Jacket.)
15	F. W. Sept. 20, 1885, vol. 130, p. 256.	36	M	Bolles.	Fell 3 stories.	Mid dorsal.	yes	no	no	Complete.	yes			yes	yes			Letter from patient Sept. 15, 1886. Can move legs slightly, but not to any practical extent; sensation fairly good; catheter constantly; scrotum and penis often œdematous; general condition improving, but bed-ridden.
16	W. C. Oct. 18, 1885, vol. 132, p. 66.	25	M	Post.	Fell 35 feet, 1 week before entrance.	Dorsal, 4th.		yes		Complete.	yes			yes	yes			Discharged incurable.
17																		(See Table for Immediate Rectification of Deformity and Fixation by P. of P. Jacket.)
18																		" " " " " " " " " " " " " "

APPENDIX.

I.

From the St. Louis Medical and Surgical Journal.

FRACTURE OF THE SPINE, WITH CASES. By T. R. WEIST, M.D., of Richmond, Indiana, March, 1880, p. 295.

ON June 25, 1879, called to see Mr. B., farmer, æt. 30, whose back, messenger said, "was broken." When accident occurred, he was on top of a load of hay, that was being driven under a shed across which some beams were placed. While passing through, he found that he was likely to be caught by the timbers above. He was sitting on the hay; he threw his body forward and lowered his head; still there was not sufficient room, and the upper part of his shoulder and spine came in contact with the obstruction above, and he was badly crushed. Was unconscious for some time after the accident. There was a serious injury and great deformity of back. A few hours later he was in great agony, complaining of violent pain in his back, and of great difficulty in breathing.

Examination:—Shock, pulse 140, feeble, skin cool and perspiring, resp. 35, difficult; deformity of spine at 9th and 10th dorsal vertebræ. On each side of the spine there was a decided swelling near the point of curvature. On shoulders, skin abraded and much bruised. Loss of sensation below seat of injury, more marked on right than on left side. No paralysis of motion. Slightest movement caused great pain. Spinous process of 9th dorsal vertebra was much displaced backwards, it being possible to place the finger under it. Evidently there was fracture of 9th dorsal vertebra, with slight dislocation backwards. Efforts were made by extension and pressure to reduce the displacement, without success. During these manœuvres distinct bony crepitation was felt. . . . . Fifteen hours after accident paralysis about the same. He was then suspended à la Sayre. I then made strong extension with slight rotatory movements, and pressure with the other hand over the projecting parts of spine. Crepitation was again felt.

Plaster jacket was now hastily but carefully applied, then he was put to bed. Breathing greatly improved; was left in comparative comfort.

Next day he was much improved; sensation below injury restored; bladder again normal. On 7th day he could turn in bed without assistance; on 12th was carried on a stretcher to his father's house 2 miles distant; on 15th day was able to get out of bed himself; on 20th day a new jacket was applied, and worn until September 1st. November 2d, 67 days after accident (?), his back is as strong as before the injury. . . . .

II.

From Centralblatt für Chirurg., Leipzig, 1880, vii. pp. 97–100.

Der Thoraxgipsverband bei Fracturen der Wirbelsäule, von Prof. König. (3 cases.)

(1) Male, æt 20. Had a fall from second story window, Aug. 14th, 1879; unconscious when found. Rallied at hospital. On examin., found a fracture of the 8th dorsal vertebra; there were no symptoms of motor paralysis, no paralysis of either bladder or rectum. On Aug. 16th the plaster of Paris jacket was applied, patient being suspended; the jacket extended from the axillæ to the trochanters. The suspension and application of bandage were well borne, and immediate improvement followed. Patient was able to walk at the end of three weeks. On the removal of bandage, about the middle of the following September, deformity had disappeared, and there were no symptoms of compression of the spinal cord.

(2) Patient, male, æt. 28. Had a fall of 40 feet, Oct. 21, 1879; was unconscious for half an hour, then complained of violent pains in back, and formication in lower extremities, with numbness; no paralysis of bladder or rectum. Back very sensitive in region of first dorsal vertebra; angular deformity at that joint.

Oct. 22d, the gypsum bandage was applied, in the same manner as in the preceding case. The nervous phenomena had disappeared by the next day, and the patient was able to move about by the 22d of November. Nov. 29th bandage was removed; the cyphosis had disappeared, and patient was dismissed perfectly well.

(3) Male, æt. 38. Had a severe fall, Nov. 28th, 1879, from roof of a rail-road car; suffered fracture of 8th, 9th and 10th rib, near vertebral insertion, with hæmo-pneumo-thorax deformity (gibbosity) in back, corresponding to the 9th and 10th dorsal

vertebra. On account of symptoms of embarrassed respiration, the correction of this deformity had to be abandoned. Cervical neuralgia. When the symptoms of embarrassed respiration had about disappeared, the gypsum corset was applied, as in the preceding cases. Nervous phenomena had disappeared the next day; patient continued to improve, and by the end of the year had perfectly recovered.

In all these cases recent fractures were dealt with, and only in such cases would I recommend the application of the gypsum corset.

III.

From Centralblatt für Chir., Leipzig, 1880, vii. pp. 737-739.

Zur Behandlung der Fracturen der Wirbelsäule mit dem Sagreé schen Gipskorsett, von W. Wagner. (2 cases.)

The author refers to the good results obtained by Prof. König in his 3 cases, and then reports two cases with which he is not satisfied:—

(1) Male, æt. 38. On Oct. 16th, 1879, was buried beneath a mass of coal, which fell upon his back; was immediately removed to hospital. There was evidently fracture of the 11th dorsal vertebra. Paralysis of bladder, which was temporary, and disappeared within 24 hours; no motor or sensory disturbances; "gibbosity," or displacement of fragments, in back. The next day patient was suspended "to toes only" (!); the "gibbosity" disappeared somewhat, but the patient complained of violent pains near the seat of fracture during the suspension. The plaster of Paris jacket was applied. During the next twenty-four hours, the pain had so greatly increased, that the patient was actually frenzied, so that the bandage had to be removed, when the pains immediately abated, and the patient afterwards recovered.

(2) Male, æt. 25. Jan. 31, 1879, sustained a great pressure while in a bent position; fracture of the 10th dorsal vertebra; no disturbance of mobility; deformity in back.

The plaster of Paris jacket was applied 24 hours after reception of injury, and was well borne at first; deformity disappeared; no increase of pain. The next day the patient complained of numbness in his lower extremities; when examined, perfect paralysis of extremities was found, also paralysis of bladder, necessitating use of catheter; decreased sensibility of extremities also found. The bandage was at once removed; all the disturbances mentioned disappeared in the course of a week. At the end of that time, patient complained of severe pains at seat of fracture when he attempted to sit up in bed. Another bandage was applied; this was worn for three months, when the patient was dismissed with slight deformity.

IV.

From Transactions of Clinical Society of London, 1881, vol. xiv. pp. 144–147.

Case of Berkeley Hill, read March 11th, 1881, before Clinical Society of London.

John Richards, on Dec. 1st, 1880, fell 20 feet down an elevator. He was unconscious until he found himself in the hospital. On his way to Univ. College Hosp. he had two fits. Never remembers having any before the accident. Suffered for 24 hours from concussion of brain and spinal cord, and was unconscious for half an hour after admitted to the hospital. Pupils dilated and insensible to light; skin hot and sweaty; pulse 120. Respiration frequent, shallow and puffing. A severe contusion behind the right parietal eminence was the only sign of injury to skull.

In the back, there was prominence of the spinous processes from the 10th dorsal to 2d lumbar vertebra; a large swelling occupied the vertebral grooves. No symptoms of visceral injury; urine drawn from bladder, normal; no paralysis of lower extremities, but great pain on movement. At 7 A.M., next day, temperature 103°; no retention of urine, no paralysis, but great pain in back. At 2 P.M., same day, the jacket (plaster of Paris) was applied; it gave great pain until it had firmly set; patient was kept until then on air-bed. The temperature then fell, patient asked for food, and then slept. Third day after application of jacket patient could roll himself in bed from side to side without pain.

Dec. 4th, patient complained of sharp pain on dorsum of left foot; no loss of sensibility anywhere. Dec. 5th, could not move the lower extremity at hip, knee or ankle, though he could flex the toes slightly. Right limb normal; but sensation of "pins and needles" in fingers of right hand. In two days control of left limb was regained, hyperæsth. of foot disappeared. Jan. 27th, jacket removed, patient allowed to walk about the ward. Feb. 2d, patient left for Eastbourne Hospital (a long railway ride). Feb. 24th, completely cured and able to walk three (3) miles.

V.

From Deutsche Z'schrift. f. Chirur., Leipzig, 1885, xxii. pp. 356, 386, by Edward de Reynier.

Fracture of 7th Dorsal Vertebra.

Patient was suspended "à la Sayre;" perfect reduction of dislocation in this position; plaster of Paris jacket was then applied, in order to insure "permanent reposition" of spine.

The injury had caused paralysis both of motion and sensation in lower extremities, which had completely disappeared eight days after application of jacket. One month later patient was able to rise and walk, with the aid of crutches. He wore the jacket for two months, at the expiration of which he left the hospital cured.

Title of article is: *Einige Bemerkungen über* 17 *Falle von Wirbelfracturen, die auf der Chir. Klinik zu Bern vom Jahre*, 1865–1884 vorgekommen sind, von EDOUARD DE REYNIER.

Patient, male, æt. 19. Admitted to hospital, Sept. 20th' 1881, for fracture of spine (exact location not mentioned); had fallen into a well head first, had his back suddenly thrust backward; states he "heard his back crack." He was then unable to rise or move and was brought to the hospital in this condition. Author regrets to say that he was only thoroughly examined five days after the infliction of the injury. Plaster jacket was not applied until Oct. 3d; the next day the sensibility and power of motion had returned, and retention of urine disappeared (no priapism).

VI.

From St. Louis Courier of Medicine, Jan. 1885, p. 71.

Meeting of "St. Louis Medico-Chirurgical Society." Dr. Carson (N. B.); "It may be interesting to the gentlemen here who were present several weeks ago, when I reported a case of fracture of the spine, to hear the result. I saw the patient shortly after the receipt of the injury, within a very few hours, and applied a plaster jacket; the patient was at that time devoid of sensibility and ability to move the extremities. I stated that immediately after the stretching he felt relieved from the pain, and all the other symptoms were also relieved; the fracture was in the lower portion of the dorsal region. The patient gradually recovered, and the bad symptoms disappeared within a few days after the application of the splint, as did the other disagreeable symptoms, and he said to me to-day that he felt as though he could go out. I don't think he could run a race, but I think he has done very well indeed.

We have had, since that time, another case, which happened very much in the same manner, and the injury is very nearly at the same site. The patient came to the hospital several days after the injury was received, in a much worse condition. We applied the jacket in this case, but not with any decided improvement, so far. The patient was unable to talk or feel the introduction of the catheter at the time he entered the hospital. At

the present time, whenever the catheter is introduced, he feels it, but there is total loss of sensation in the lower extremities, and his condition is not a favorable one, nor has there been any material benefit by the application of the splint. In this case, however, we did not suspend him as we did in the other; we tried extension and counter-extension from the hip and shoulder in the horizontal position."

VII.

W. L., æt. about 50. Carpenter, strong and vigorous. Fell from staging, about thirty feet, May 21, 1881.

Was doubled up and taken home in a hack. I saw him soon after. Fracture of spine near middle dorsal. Paralysis complete. Priapism, retention of urine. Motion of fragments marked at point of injury. After a careful explanation of the danger, extension was made and a plaster splint applied, aided by Dr. Samuel N. Nelson. For a few days there was a marked improvement, both sensation and motion in a slight degree returning to both extremities. Soon the patient grew worse, with rapid pulse, elevation of temperature, delirium, sinking into a coma, and death supervened June 4th, fourteen days after the injury.

Autopsy showed a transverse fracture through the body of the vertebra with local softening of the cord at the place of injury. The fragments were in direct apposition.

Case 2d.—P. R., horse doctor, æt. about 50. In good general health, except suffering from multiple strictures. Had been a hard drinker.

In August of 1882, was thrown backward from carriage, striking violently on shoulders. Paralysis not complete. Sensation and motion of lower extremities, to a limited extent; retention of urine. Not seen for some days after injury, owing to absence from city; under care of another physician. Was a distinct projection with marked tenderness over the second and third dorsal vertebræ, with slight motion on firm pressure. In consultation with Dr. F. A. Holt, of Cambridge, it was determined to attempt extension and reduction, after a careful explanation, of the condition and its danger, to the patient.

Upon extension a certain amount of displacement was effected and a plaster splint applied. The patient fainted, with entire loss of consciousness at its completion, but rallied at once upon being placed horizontal on a water bed near at hand. Owing to pressure and disturbed respiration the splint was carefully opened anteriorly, and retained by tapes. Was kept on water bed three

weeks, and then carefully removed to a firm hair mattress. Paralysis slowly lessened, until patient could walk, at the end of about six months. Now is able to attend to business. Can stand upon either foot and has no pain or tenderness at seat of injury. The patient is stooped, with a considerable prominence at site of injury, and carries the head somewhat forward.

THE ARTIFICIAL FEEDING OF INFANTS.*

BY T. M. ROTCH, M.D.,

Visiting Physician to the Boston City Hospital; Instructor in Diseases of Children in the Harvard Medical School.

WHILE recognizing the importance of feeding infants during the early months of life by means of human milk, we must allow that in civilized communities the necessity of supplying the infant with food not directly from the human breast must often arise, and will in all probability be a demand which will increase rather than decrease as our civilization advances; and when in addition to this we consider the great proportionate mortality of the artificially-fed over the breast-fed, and the difficulties which are so frequently met with in adapting the food to the individual case, it manifestly becomes a duty to carefully investigate the different methods of artificial feeding, and adopt some more uniform plan for starting human beings in life than is met with among physicians as a class and the laity as a whole, for diversity and not uniformity is the rule.

With the exception of the very small proportionate per cent. of inherited diseases which occur at birth, this diversity of method in feeding is the most prolific source of disease in early infancy. The group of symptoms which for want of a better name is represented by dyspepsia, difficult digestion, occurs most frequently in the three periods when the infant's digestion is likely to be tampered with,—namely, in the early weeks of life, when experiments are being made to determine what food will be best to start with; next, when in addition to the irritation arising from the beginning of dentition new articles of diet are added to the original food; and, thirdly, at

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* Read before the Obstetrical Society of Boston, May 28, 1887.

the time of weaning, when there is often a sudden and entire change in the character of the food.

The proper management of the first of these periods is of the greatest comparative importance, because it is the time when the function of digestion is being established and is in a state of unstable equilibrium, and therefore, following the rule of functional establishment, the stomach is in its most active period of growth, and hence the most careful regulation of the bulk of the food given is needed to correspond to this activity in order that we should not weaken the digestive function by overtaxing its capacity and yet provide the proper materials for nutrition, thus avoiding by prophylaxis the dyspepsia of the later periods of infancy and childhood, the seeds of which are continually being sown in this early transitional period.

We therefore have the question not only of infantile digestion but infantile development to deal with, and we should at once recognize the fact that the problem of artificial feeding is not a simple factor,—namely, which food shall we give to the infant, —but is a combination of factors, of which the kind of food is only one, and all these factors, from which we deduce the general problem for the average infant and the especial problem for the individual, must approach as closely as possible to the analogous factors which nature freely presents to us for investigation,—that is, we must follow nature as closely as possible. Our scientific knowledge and ingenuity have not yet enabled us to follow nature exactly, and we therefore have not yet obtained an ideal method of artificial feeding; but we must, nevertheless, go as far as the present state of our knowledge will allow, thus gaining a little ground every year; and we must be especially careful not to be led astray by the fictitiously brilliant results which are reported from time to time in favor of certain foods, instances continually occurring where one food will fail and another when substituted for it succeed; and yet these successes are merely temporary, and the mortality always remains far above that of human breast-milk.

It is certainly wiser and more economical not to spare expense and trouble in arranging the infant's diet, for, as has been explained above, the period of active growth of an organ

is the time when its function is readily weakened, and when once weakened the digestive function is a prolific source of annoyance and expense in childhood and adolescence. Cheap foods and cheap methods of feeding, unless they are the best that can be procured, should no more be tolerated, and in fact not nearly so much so, in the early feeding of infants than in adult life: we often, however, see a food recommended for a young infant because it is cheap and easily prepared, and yet where its well-known lack of nutritive ingredients would with adults at once stamp it as unfit for use.

What are the general factors of the problem which constitutes nature's method of feeding? We have first a receptacle, the human breast, which mechanically provides a fresh supply of food at proper intervals, absolutely prevents fermentation of the food before it enters the infant's mouth, forms the mouth by the process of sucking, incites to action the necessary digestive fluids, avoids a vacuum by collapsing as it is gradually emptied, thus allowing the food to flow continuously, and finally is practically self-regulating as to the amount of daily food according to the infant's age. Second, the food itself adapted to the infant's digestive function and for its development, by its temperature, 98° to 100° F., its alkaline reaction, and its chemical constituents. Given these factors, how nearly can we approach them artificially? Human ingenuity has not yet been able to devise anything which approaches the perfection of nature's receptacle, and the best that we can do to offset this complex mechanism is to adopt that which is exactly the reverse,—namely, a receptacle of absolute simplicity,—and thus combat the tendency to fermentation by, through perfect cleanliness, preventing the receptacle from becoming a source of fermentation. To meet this demand I have had made what are practically enlarged test-tubes, which not having any angles are readily cleaned.

The receptacle, however, has to receive a food which usually is non-sterilized, and hence, where the factor of fermentation appears to be prominent in disturbing the infant's digestion, the food should be sterilized before it is given to the infant.

The process of sterilization is most simply accomplished by placing the food in one of the above spoken of feeding-tubes,

adjusting the rubber nipple as on any nursing-bottle, then drawing tightly over the nipple and well down on to the tube a strong rubber cot, which, not being perforated, completely excludes the air. The tube is then exposed to steam confined in any vessel for twenty minutes, which is sufficient to render it sterile, in the sense of destroying the developed bacteria and thus making it correspond to human milk; at the same time, so far as my investigations on this subject have gone, the steam, while it sterilizes, does not apparently alter the chemical attributes of the food, as is essentially the case where the sterilization is accomplished by boiling; then again, in the steaming process the receptacle is sterilized as well as the food, so that when the rubber cot is removed we have the food enter the infant's mouth as free from bacteria as has been shown to be the case with human milk by Esherich,* who experimented with the milk of twenty-five healthy women, and found, by keeping it in sterilized tubes, that it remained sterile for some weeks, while, on the contrary, in women whose temperature was raised from fissures and excoriations of the nipple and by general puerperal infection, bacteria were found in abundance. It is thus seen that this factor of sterilization is likely to be found of considerable importance in the future, and should certainly be called to our aid in those cases where the other factors of our problem are as nearly correct as we can make them, and, if for no other reason, because nature plainly tells us that a perfect food should be sterile, and, in fact, our practical clinical experience for years has taught us to withdraw the infant from the breast in exactly that class of diseases where we now know bacteria to occur in the milk. Reference has been made above to the developed bacteria; if it is desired to prepare the food so that it shall remain sterile for some time, it is necessary to sterilize for several days in succession, for the first sterilization, according to Dr. Harold Ernst, Demonstrator of Bacteriology in the Harvard Medical School, only destroys these developed bacteria, while the spores are left to develop later. Dr. Ernst also kindly placed one of my tubes in which there was a mixture of cream, milk, lime-

* Baumgarten's Jahresbericht. Erster Jahrgang, 1885, p. 34.

water, and milk sugar in his sterilizer for twenty minutes and then in his incubator for twenty-four hours, and at the end of this time no change could be detected in the mixture, either in color, odor, or taste, all of which appeared to exactly correspond to a freshly prepared mixture of the same kind.

I have devised a simple apparatus for household use, which can be made at any tinsmith's at small expense, and answers for the purpose of sterilization very well.

It is a round tin pail, eight inches in diameter and fourteen inches deep, raised on three legs, sufficiently high to allow an alcohol lamp to stand under it; four inches from the bottom of the pail on the inside is a perforated tin diaphragm on which the feeding-tubes stand while being sterilized.

The pail has a cover and handle. Water is placed in the bottom of the pail, and when heated by the lamp the tubes are soon enveloped in steam.

The process of sucking is accomplished by the rubber nipple as by the breast, and a small hole near the end of the feeding-tube prevents a vacuum being formed and regulates the rapidity of the flow, while it allows it to be continuous; this is done by rolling up the edge of the rubber nipple from the hole with the finger, or letting it cover the hole according to the demand shown by the infant when feeding.

The artificial receptacle is not self-regulating, and hence we must first determine anatomically the amount of food in bulk which nature provides for the average infant at different ages, and from these average figures deduce the proper amount for the especial infant, having also the feeding-tubes graduated for the more important periods of growth, for the purpose of continually impressing, upon the mother and nurse, what the physician only has the opportunity of telling them at the beginning of the nursing period,—namely, that the error is in giving too much food rather than too little, an error also which naturally results when, as is commonly the case, the usual eight-ounce nursing-bottle is provided as the receptacle at the very beginning of infantile life.

Diagram I. represents the stomach of an infant five days old, in life-size.

The specimen was prepared by Dr. C. W. Townsend, and was found to hold twenty-five cubic centimetres.

DIAGRAM I. DIAGRAM II.

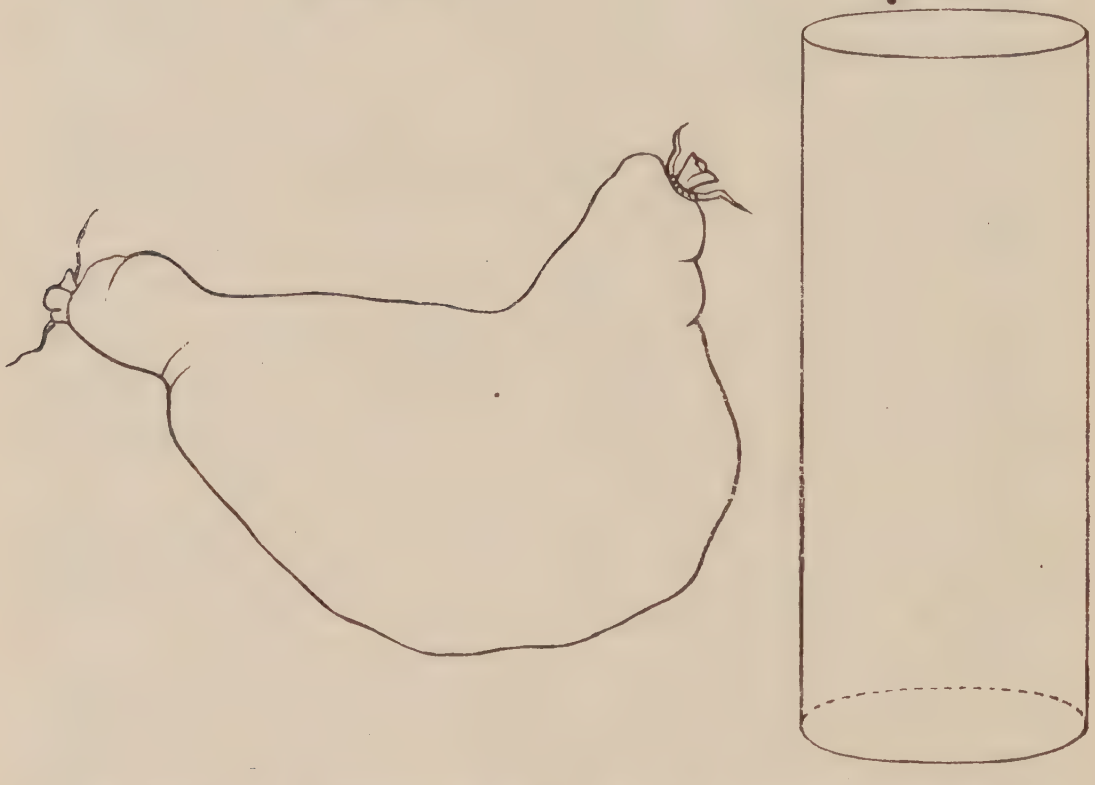


Diagram II. represents the size of the tube, which is sufficiently large for each feeding during the first week; and when we consider the space which would be needed to represent the full-sized nursing-bottle, these two diagrams express better than can be explained by words the disproportion between the size of the infant's stomach and the amount which the mother supposes it should hold to keep her child from being starved.

Frolowsky's* investigations show that the activity of growth in the stomach's capacity can be represented by the ratio of one for the first week, to two and one-half for the fourth week, and three and one-fifth for the eighth week, while it is only three and one-third for the twelfth week, three and four-sevenths for the sixteenth week, and three and three-fifths for the twentieth week.

We thus see that there is a very rapid increase in capacity in the first two months of life, while in the third, fourth, and

* Inauguraldiss., St. Petersburg, 1876.

fifth months the increase is slight. Guided by these data, which we find correspond closely with the results of clinical investigations bearing on this point, we should rapidly increase the quantity of the food in the first six or eight weeks, and then give the same quantity up to the fifth or sixth month, unless the infant's appetite evidently demands more, when of course a gradual increase should be made. A considerable increase in the quantity needed, also, usually takes place between the sixth and tenth month.

Ssnitkin, after a series of careful investigations in the Children's Hospital at St. Petersburg to determine the amount which should be given in the first thirty days of life, finds that the greater the weight the greater is the gastric capacity. His general results also show that one one-hundredth of the initial weight should be taken as the starting figure, and to this should be added one gramme to each day of life. This, for example, gives the following amounts for each feeding, which closely correspond to the average figures which I have computed and introduced in Table I. on the basis of three thousand grammes as the initial weight. Thus, one one-hundredth of three thousand would be thirty grammes for the early days, and at thirty days the amount given would be $30 + 30 = 60$ grammes, about two ounces.

The younger the infant the greater the metabolic activity, and hence the greater need for frequent feeding, for nutriment is required not only for the excess of waste but also for the rapid proportionate growth; this makes the intervals of feeding a factor of considerable importance in the management of the infant's diet.

The figures in Table I. are merely approximate average computations taken from the results of a number of Russian, German, and American observers and from my own experience during the past ten years, and are only intended to be a guide for the physician in his management of cases of difficult digestion, for of course some infants will have a greater appetite and a greater power for digestion than others of the same age and weight.

Table I.

The average initial weight of infants is 3000–4000 grammes = about 6–8 pounds.
The average normal gain per day in the first 5 months is 20–30 grammes = about 1 ounce.

General Rules for Feeding.

Age.	Intervals of Feeding.	Number of Feedings in 24 Hours.	Average Amount at each Feeding.	Average Amount in 24 Hours.
1st week .	2 hours.	10	1 ounce.	10 ounces.
1–6 weeks .	2½ hours.	8	1½ to 2 ounces.	12 to 16 ounces.
6–12 weeks, and possibly to 5th or 6th month . .	3 hours.	6	3 to 4 ounces.	18 to 24 ounces.
At 6 mos. .	3 hours.	6	6 ounces.	36 ounces.
At 10 mos. .	3 hours.	5	8 ounces.	40 ounces.

The weight, as well as the age, is necessary to determine the amount for each feeding in the individual infant, the rule being $\frac{1}{100}$ of the initial weight + 1 gramme for each day during the first month.

Illustrations of above rule to serve as guides for especially difficult cases.

Initial Weight.	Each Feeding. Early Days.	At 15 Days.	At 30 Days.
3000 grammes.	30 grammes (about ℥1).	30 + 15 = 45 grammes (about ℥1½).	30 + 30 = 60 grammes (about ℥2).
4500 grammes.	45 grammes (about ℥1½).	45 + 15 = 60 grammes (about ℥2).	45 + 30 = 75 grammes (about ℥2½).
6000 grammes.	60 grammes (about ℥2).	60 + 15 = 75 grammes (about ℥2½).	60 + 30 = 90 grammes (about ℥3).

I have experimented with the glass-blower in having tubes of different sizes made, and finally out of a large number of samples the following have been chosen as the most practical and the easiest to handle. For convenience I have had these tubes fitted to a box, which also contains directions for their

use. The tubes as seen in Diagrams III. and IV. are merely enlarged test-tubes blown into a glass standard, and having a small hole at the mouth for the entrance of air.

DIAGRAM III.

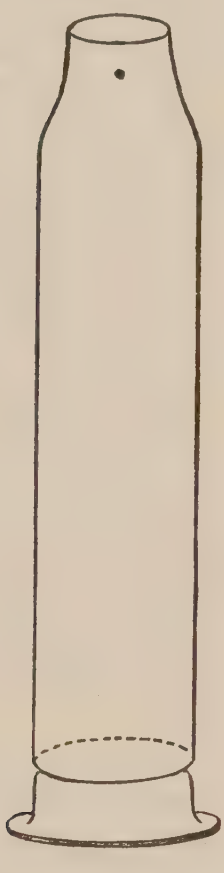
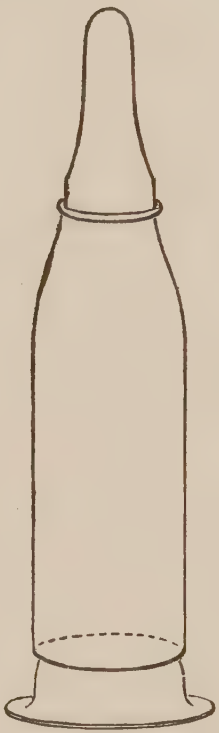


DIAGRAM IV.



A measuring-glass graduated to hold two ounces, and shaped like the larger tubes, is also in the box, and is used as a feeding-tube during the first six weeks, and later as a measure for the larger tubes. The smaller tube, Diagram IV., holds four ounces, has a calibre of one and five-eighths inches, and a height of six inches; it is to be used from the sixth week to the fifth or sixth month, and is intended to correspond to the above-described rapid growth of the stomach in the first two months, and its insignificant further increase in size up to the fifth or sixth month; it is represented in the diagram with the nipple adjusted for use. The large tube, Diagram III., has a calibre of one and six-eighths inches, a height of eight and three-fourths inches, and corresponds to the common half-pint nursing-bottle; it is represented in the diagram without the nipple, and shows the air-hole.

The box, besides the rubber nipple, contains a rubber cot

for sterilization, test-paper for ascertaining the reaction of the food, and a bottle of soda for keeping the tubes pure during the intervals of nursing.

A medium-sized tube, which does not come with the box, but can be had separately, has a calibre of one and six-eighths inches, a height of seven and three-fourths inches, and holds six ounces. It of course is not a necessity, but is intended to be used between the sixth and tenth month, merely to enunciate the importance of careful supervision of quantity throughout the first year, as where a food qualitatively correct is being used, the error, as a rule, is in giving too great an amount quantitatively than too little, and a food which otherwise would be digested perfectly well often fails if it is given in too large an amount or at improper intervals; we may, however, here notice that while in breast-feeding frequent feeding is contraindicated from its altering the character of the food, in fact, condensing it, this trouble does not of course arise in artificial feeding, the food remaining the same, and we thus have a little more latitude given us in our management than we can have in regulating the breast-feeding.

The question of the food itself in artificial feeding is practically reduced to some modification of cow's milk or to cow's milk itself, for in most civilized communities cow's milk is far more easily obtained than other milks; and although the milk of other animals may approach more nearly the per cents. of the ingredients of human milk, they must all be modified, and if we modify at all, it is as easy to deal with cow's milk as with these other milks. Then, again, the various foods, patent or not, all depend for their basis on milk, and without this addition of milk would show but an insignificant percentage of many of the most important ingredients of the food, so that logically we should speak not of the various foods as such, but merely as adjuvants to cow's milk, for if this is thoroughly understood it will, in many minds, do away with much misapprehension regarding the apparently successful results of innumerable foods which, in reality, when given to the infant, are merely a means of modifying the almost universal representative of the artificial foods, cow's milk. Cow's milk, therefore, should be carefully compared with the standard

human milk in order that we should know how nearly it resembles it, and Table II. is a comparison of the two foods, the figures representing the later and more reliable analyses. In considering the preparation of the various foods with reference to making them correspond in their analyses as nearly as possible to human milk, the problem is somewhat simplified if we recognize the fact that although the per cents. of the ingredients of human milk vary under certain circumstances, yet so far as the age is concerned, in the early months there is so little variation that a variation is as likely to occur between different milks of the same age as the same milk at different ages, so that we probably are doing wisely not to change the per cent. of the ingredients, but as the infant grows older give a food qualitatively uniform but of varying quantity.

From the comparison, we at once see that cow's milk itself differs so markedly from human milk that it evidently should be modified before being used as a food.

It may be modified by water, or by the patent foods, or by any adjuvant, such as barley-water, lime-water, or its own cream.

There is a very large number of patent foods, but they all claim about the same advantages, and closely resemble each other in their constituents, and in their honest endeavor to make cow's milk easily digestible, and also to make their resulting analysis agree as closely as possible with human milk. There are, however, certain differences by which we can divide them into classes, and we can speak of individual foods as representing their class.

Table II.

Human Milk.		*Cow's Milk.*
87–88	Water	86–87
13–12	Total solids	14–13
4.	Fat	4.
1.	Albuminoids	4.
7.	Sugar	4.5
0.2	Ash	0.7
Alkaline.	Reaction	Acid.
Small proportionately	Coagulable albuminoids	Large proportionately.
Scarcely perceptible in test-tube.	Coagulation by acids	Marked in test-tube: greatest with pure milk; least when mixed with lime-water or barley-water.
Not present.	Bacteria	Present.

My own opinion in regard to patent foods as a whole is that they must necessarily be unreliable; they are thrown on the market in such numbers that the competition is extreme, and when once they have made a reputation I cannot but feel that irregularities and changes, slight, perhaps, in the eyes of the makers, may unintentionally creep in and carry their analysis still further from that of the standard human milk than it was in the beginning.

Analyses show that there is a lack of uniformity from year to year, and that original claims are apparently forgotten or allowed to give way to cheaper production. A striking example of the truth of this statement is the world-wide reputation of Mellin's Food, with its printed analysis, by Dr. Leeds, showing that it contains no starch, its starch having been converted into sugar; and yet careful analyses made later by Dr. Charles Harrington, of the Harvard Medical School, show conclusively that all the starch has not been converted into sugar, and that it is present in very appreciable quantity,—perhaps, as it may be argued, not in sufficient quantity to do any harm, but, as it is claimed not to be present at all, it gives rise to an element of uncertainty. If we wish to be exact in the preparation of our food, and if we wish to introduce starch into the food, we should prefer to use a food where starch is acknowledged to exist, and can be reduced to the amount which we may deem necessary for the especial case; and yet just as the makers of Mellin's Food in good faith state that it contains no starch, while the unquestioned reputation of Dr. Harrington as a chemist proves that in certain cases at least they are mistaken, so in the patent foods which claim to contain starch in a certain percentage, that percentage may vary so much as to make any combination which we may wish to make an uncertain rather than an exact one. Besides the objections just given to these foods as a whole, it will be seen by referring to the representatives of each class as shown in Table III. (page 14), that, even taking the analysis as given by Dr. Leeds and also the percentage of the ingredients when mixed, according to the maker's direction, with cow's milk, there is a striking similarity in all of them, in that they all show a marked variation from the standard human milk.

The patent foods can practically be divided into those which are manufactured from cereals and those which are not. The first class contains the starch of the cereal unchanged or converted into sugar.

We have then, I. A cereal food with its starch unchanged, represented by Imperial Granum. II. A cereal food with its starch claimed to be converted into sugar, represented by Mellin's Food. III. A cereal mixed with condensed milk, its starch unchanged, and represented by Nestle's Food. IV. Condensed milk. V. The so-called humanized or peptonized milk.

I have prepared Table III. not only to show the ingredients of the representative foods (the analyses of the patent foods in Table III. were made by Prof. Leeds, and were published in the *Medical News* of July 21, 1883), but the ingredients of the food as it is usually given to the infant, mixed with cow's milk, and reference to this table and to the analysis of human milk on the upper line will not only show the errors of nutrition, which are made when we use them in this way, but enables us, by a simple process of calculation, to make any of them correspond more closely to the standard, for all of these foods are at times useful, provided that we use them intelligently for the especial case; but, of the foods represented in the table, we should avoid the patent foods for general use on account of the objection referred to above regarding their manufacture as a class; they are also the most difficult class of foods to make correspond to human milk.

An examination of Table III. will at once explain how difficult it is to make the artificial foods correspond to human milk by the methods which are usually employed, and also the errors in percentage which result from these methods; it is, in fact, a series of figures which represent the element of nutrition rather than digestion, and the merits of every food should be determined in this way before submitting it to the test of clinical experience, for our common sense must certainly be better satisfied if we know that the infant is not only digesting the food, but that the food itself is similar (or as nearly so as we can make it) in its proportions, ingredients, and reaction to the standard which, in its results, shows the lowest rate of mortality.

Table III.

Comparison of Human Milk with Cow's Milk and Cow's Milk Modified.

(The figures are approximate and represent general averages.)

	Reaction.	Starch.	Water.	Total Solids.	Fat.	Albuminoids.	Sugar.	Ash.
Human milk	Alkaline.	0	88	12	3–4	1–2	7	0.1–2
Cow's milk	Acid.	0	87	13	4	4	4.5	0.7
Cow's milk 1 Water 2 3	Acid.	0	95.61	4.39	1.33	1.33	1.5	0.23
Cow's milk 1 Water 1 2	Acid.	0	93.4	6.6	2	2	2.25	0.35
Condensed milk	....	0	28	72	10	10	50	2
Condensed milk 1 Water 9 10	Neutral.	0	92.8	7.2	1	1	5	0.2
Condensed milk 1 Water 6 7	Neutral.	0	89.66	10.34	1.45	1.45	7.14	0.3

Imperial Granum. Fat 1.01 Albuminoids 10.51 Sugar trace. Ash 1.16 Starch 78.93 — 3 Milk 32 Water 64 99	Acid.	2.36	92.88	7.12	1.33 .03 1.36	1.33 .31 1.64	1.5 trace. 1.5	0.23 0.03 0.26
Mellin's Food. Fat 0.15 Albuminoids 5.95 Sugar 48.2 Ash 1.89 Starch present. — 3 Milk 48 Water 48 99	Acid.	Present.	91.74	8.26	2.0 .004 2.004	2.0 .17 2.17	2.25 1.44 3.69	0.35 0 05 0.4
Nestle's Food. Fat 1.91 Albuminoids 8.23 Sugar 38.92 Ash 1.59 Starch 40.1 — 1 Water 10 11	Neutral.	3.63	91.69	8.31	0.17	0.74	3 63	0.14
Barley-Water. As usually made with Robinson's barley contains *starch* 1.4. — 1 Milk 2 3	Faintly acid.	0.47	90.75	9.25	2.66	2.66	3.0	0.46
* Cream 2 Milk 1 Lime-water ~~1~~ † Sugar-water 3 8	Alkaline.	0	88.42	11.58	4.0	1.11	6 26	0.21

* The cream must contain from 14–16 per cent. fat.

† The sugar-water consists of milk-sugar 5 17¾; water 01.

It will at once be seen that no matter how cow's milk is diluted with water it cannot be made to correspond to human milk. It is well, however, to remember that clinical experience has shown that infants seem, even in the early days of life, to digest the casein well enough, provided that it is sufficiently diluted,—that is, about four times, which reduces it to one per cent., and this will be of significance when we come to prepare a food which will correspond to human milk. If, however, we reduce cow's milk so that the percentage of albuminoids is one per cent., the fat and sugar fall so far below the standard that, although the ash has the proper percentage, yet we have an acid food markedly deficient in its nutritive quality.

Condensed milk is one of the most interesting foods which we are called upon to deal with, and represents in its production, its chemistry, and its clinical results an almost perfect illustration of all that has been so far said on the subject of artificial foods: it has strong advocates and strong opponents, but a simple consideration of its vital properties will easily explain the causes of its successes and of its failures.

The process of manufacture of condensed milk sterilizes it to a certain degree, and we thus have a very important factor in its favor which does not exist in cow's milk: it is also superior to cow's milk in that when mixed with water it is, although not alkaline, still not acid, and its large percentage of cane sugar helps to avoid the occurrence of fermentation, which so readily occurs in cow's milk. In Table III. the percentage of the ingredients of condensed milk—when diluted, as it commonly is, ten times—is given, and we at once see why it is easily digested but non-nutritious, for two of its ingredients, notably the fat, are found to have fallen below the standard as represented in the upper line of the table, the ash and casein only attaining the proper percentage. The nearest approach to the standard is obtained by diluting it with six parts of water, which, as shown in the table, results in giving the proper per cent. for the albuminoids, sugar, and ash, but the fat is still much reduced, and, unless supplied in some way, we should suppose that the nutrition would suffer, and this supposition appears to be supported by clinical results. That is, clinically, condensed milk represents a food easily digested but not suffi-

ciently nutritious: the former explained by its low percentage of albuminoids and ash, its neutral reaction, its anti-ferment properties, and its proper per cent. of sugar; the latter by its great lack of fat. Among the poorer classes and in infant asylums it is a favorite food for the physician to prescribe, because the infants digest it so easily, but the testimony of those clinical observers who look beyond the temporary digestion to the subsequent nutrition of the child supports the view that condensed milk, even if we set aside the objections which, in general, arise from its being a patent food, must be modified by more than the addition of water before it can safely be given as a continuous food to the average infant. For preparing the way for other more nutritious foods in cases of difficult digestion, for convenience in travelling, and where for any reason the intelligence or the proper desire to take trouble about the food is lacking in the parents, condensed milk, from its simplicity in preparation as well as from its other attributes mentioned above, is a valuable addition to other more rational methods of artificial feeding. The commonly accepted opinion that condensed milk contains too much sugar is an error, for by referring to the table it will be seen that, as usually given, the sugar in the mixture is below the proper percentage, but if it is diluted six times, as recommended above, we then have merely the fat to deal with, and the reaction, which should be made alkaline. We must, then, modify this condensed-milk mixture; and not only is the fat an important factor in this modification, but the proper amount of fat: for although it is admitted that a large per cent. of surplus fat is frequently found in the napkins of infants whose digestion and nutrition are normal, and whose food is breast-milk, yet we have no right to conclude from this that a small per cent. of fat is sufficient for nutrition or that a large per cent. will be taken care of by this outlet; in fact, it is far more probable that nature introduces a certain per cent. of fat in human milk with a purpose which can only be accomplished by that per cent., and that it is an error to vary this per cent. beyond the variation which commonly occurs in average human milk. From what has been said above we would naturally expect that unless the standard per cent. was attained, or at least a near approach to it, trouble

would be likely to arise, and this corresponds to my individual experience in the cases where the especial ingredient which was disturbing the success of the food was the fat: for I have found clinically that under the proper per cent. of fat the nutrition suffers, and that where the fat percentage was decidedly above the standard both nutrition and digestion were affected unfavorably. Unless, then, it is impossible to be more exact in arranging the percentage of fat in condensed milk, as is often the case among the poorer classes, where cod-liver oil is used as a cheap expedient for rectifying this source of error, the addition of indefinite amounts of fat to a food is to be deprecated, just as it is unwise to add indefinite amounts of sugar, and we should seek for a better combination than is offered to us in condensed milk.

Any food which introduces an element foreign to the ingredients of human milk is to be looked upon with suspicion, as it is not likely that we can improve on nature's method of adapting the food to the infant's digestive functions; we should therefore consider carefully before recommending the various classes of food which contain starch, which by referring to Table III. will be seen to be the foreign element which enters into the representative patent foods, Imperial Granum, Mellin's Food, and Nestle's Food.

It of course is not merely necessary to know the percentage of the different ingredients as they exist in the food itself, for what concerns us is the percentage as given to the infant, and the table therefore shows the sum totals of the milk percentages and the food percentages, and a glance at these figures tells us whether we have an easily digestible and nutritious food to deal with.

For instance, the success of Imperial Granum is evidently in its correct percentage of albuminoids and ash making it easily digestible, but its failures are readily explained by its reaction, its foreign ingredient, and its very low percentage of fat and sugar.

Reference has been made above to the capability shown by even very young infants to digest the casein of cow's milk when it is reduced to one or two per cent., and this is a factor which probably enters to a greater degree than is usually

recognized in the easy digestion of these foods, and possibly too much credit has been given to the starch as a means of making the casein more digestible.

This was suggested by Dr. Arthur V. Meigs some years ago, and is worth considering, as it certainly is more rational not to introduce a foreign ingredient like starch into the food if we can make it digestible in some other way. Examining this question of casein percentage in the three large classes of patent foods represented in Table III., we find Imperial Granum 1.64, Mellin's Food 2.17, and Nestle's Food .74. Now all these correspond very closely to the casein percentage of human milk, that of Mellin's Food perhaps being rather high, and all these foods are found to be easily digestible; so also where barley-water is mixed in the usual way with milk, one to two, the casein of the resulting mixture is notably diminished in amount and naturally is more easily digested than when it stands at a higher per cent., as it does in cow's milk undiluted. The fat and sugar also are very notably diminished in all these mixtures. We therefore find from the table that there is a decided failure to fulfil the factor of nutrition when cow's milk is modified with these foods, and that all of them need still further modifications, some in one ingredient and some in another, that they all contain a foreign ingredient, and all, when mixed and prepared for use, fail in that important requisite for at least the early months of life, an alkaline reaction.

There are, of course, many other foods which might be spoken of, but they all come under one or the other classifications above referred to. It may be well, however, to say a few words about peptonized milk. This is cow's milk with its casein partially or entirely predigested by means of the extract of pancreas and soda. Now there is no doubt but that the casein of cow's milk has been a source of trouble to the infant's digestive apparatus, and under certain circumstances can, with great benefit to the infant's digestion, be treated by predigesting it for a time and thus allowing a stomach, which otherwise digests well, to rest and recover itself. It is of use, also, where a decided idiosyncrasy of the individual precludes the digestion of this especial ingredient of the milk; but besides the probability that the indigestion is often attributed to

the lack of power to digest casein at all, while in fact the stomach is simply rebelling against an amount of casein above the standard per cent. or against some of the many other factors of the problem, it would seem that for the average infant digestion this predigesting of the casein or any other constituent of the milk is contrary to nature's teaching. There are certain natural functions which should be allowed to act as they do on human milk, and it seems irrational and contrary to the laws of physiology not to encourage all the functions to act naturally, each in its own province, instead of forestalling their action and allowing them to fall into disuse and thus be weakened. The baby's stomach is intended to digest casein, and not to have the casein digested for it. Clinically, also, the use of peptonized milk supports this view, for, so far as I know, no very brilliant results have been obtained from its use. Peptonized milk, then, is a food consisting of too large an amount of digested casein, too little sugar, and a very large over-proportion of ash.

The question next arises as to whether cow's milk can be modified without the use of patent foods or foreign ingredients and made to correspond to the percentages given in the upper line of Table III. This has been accomplished, with more or less success, by the addition of fat in the form of cream and by alkalies. It seems to me, however, that unqualified credit should be given to Dr. Arthur V. Meigs, of Philadelphia, not merely for devising the mixture which will be spoken of presently, for any good mathematician and chemist could have done that, but for carrying out the principle which it has been one of the chief objects of this paper to inculcate,—namely, to prepare the food free from foreign ingredients and, by chemically altering the constituents of cow's milk and especially reducing the casein to one per cent., to clinically adapt the food to the average infant's digestion. Common herd cow's milk can be diluted until the casein is reduced to one per cent., the reduced fat is then raised again to the standard by the addition of cream of a known per cent., and the sugar is also brought up to the per cent. found in human milk by means of sugar of milk. The ash is reduced to the proper per cent. at the same time that the casein is, for if we

divide the average per cent. of albuminoids and of ash in cow's milk by four, we obtain the per cents. of albuminoids and ash as they are found in average human breast-milk.

It will be noticed that the figures in Table III. give the per cent. of the ash as a whole, although we know that a number of different ingredients make up the analysis of this portion of the milk. In the future, chemistry will in all probability give us exact data regarding this analysis of the ash, and we can then by computation make a corresponding combination in the artificial food. At present, however, we are forced from want of sufficiently accurate knowledge on this point to compute the ash as a whole. Empirically, Dr. Meigs has shown that by combining two parts of cream containing from fourteen to sixteen per cent. of fat, one part average milk, two parts lime-water, and three parts sugar-water, the latter consisting of seventeen and three-fourths drachms of milk-sugar dissolved in one pint of water, we have an alkaline mixture with the percentage of its ingredients closely corresponding to that of human milk.

The figures in Table III. are the result of an analysis made by Dr. Charles Harrington of a mixture made as directed above, and the similarity to the figures in the upper line of the table representing the analysis of human milk is very striking. A number of analyses have been made for me by Dr. Harrington at different times and with different creams, and they have all corresponded closely to this one, provided that an analysis of the cream was first made in order that the proper per cent. of fat should be added to the mixture.

To avoid the necessity of having the cream analyzed, cream made by the centrifugal process can be used, for this has a per cent. which varies very little from thirty-two, so that by diluting this cream with water one-half we have a cream with a per cent. of about sixteen, which is what is usually needed for the average infant digestion.

It is possible, then, in artificial feeding, to approach the standard human breast-milk much more nearly than is usually attempted, and there is no reason why clinical results should not be much improved if physicians will only take additional time and trouble to follow more uniformly nature's teaching.

In all classes of life a much greater amount of time, expense, and thought is given proportionately to the preparation of food for the adults of the family than for the infants, and this is a mistake both from a humanitarian and an economical point of view, for the infant is much more susceptible to irregularities of diet, with their resulting suffering, than the adult, and when once the train of symptoms usually called dyspeptic is established, infinitely more trouble and expense is entailed than if more exact methods of feeding were adopted before the digestion has been tampered with. In the early weeks of lactation, after the mammary function has been fully established, it is well to have a number of analyses made of the mother's milk, and to keep the results as a control record to act as a guide for the preparation of an artificial food in case, as so frequently happens, something should occur to end the nursing at an early period, for it is highly probable that the digestive function of the individual infant may have certain idiosyncrasies which correspond to some idiosyncrasy in the percentages of its mother's milk, and in cases of difficult digestion where the artificial food, which has been made to correspond to the analysis of average woman's milk, fails to agree, reference to this control record may accomplish the solution of the problem sooner than if we had to ascertain experimentally, by changing in turn the percentages of the different ingredients, in which ingredient the idiosyncrasy of this especial infant was to be found. The assistance of the skilled chemist is too little sought after in determining these questions of infantile digestion and nutrition, and in the future must necessarily be made use of if there is to be any advance for the better in this subject of artificial feeding.

Many instructive cases could be cited, and must occur in the practice of every physician, illustrating the importance of not omitting the careful regulation of every factor of the problem, when we attempt to substitute an artificial food for the natural method of feeding, and proving that no one factor can be given undue prominence at the expense of the others, without in some way causing disturbance to the digestion and general nutrition. Making the food alkaline will at times rectify an

otherwise intractable digestion: again, as in a case of Dr. Ernst's, sterilization was the only change that was needed.

I have a number of cases showing the importance of a precise regulation of the fat percentage, and also of the sugar and the casein, while, many times, the food, although chemically correct, has proved a failure because it was given in improper quantity or at irregular intervals.

Where an infant, then, is to be fed with artificial food, give precise directions as to the times of feeding, the amount at each feeding, and the feeding apparatus which is to be used. See that the analysis of the food corresponds as closely as possible to that of human milk; give instructions as to the proper temperature of the food; see that the reaction is alkaline, and then if there is any difficulty with the digestion, sterilize the food. If this is not successful, refer to the control record and adapt the food to any maternal idiosyncrasy shown by this record. If no control record has been kept, experimentally try to discover the especial idiosyncrasy of the individual infant by changing the per cent. of the fat, sugar, casein, or ash.

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AN

EXPERIMENTAL RESEARCH UPON RABIES.

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Pasteur's work upon and claims in regard to rabies are so well known, more particularly the latter, that any detailed account of them would be superfluous here. Starting with material obtained from a child dead of supposed rabies, in one of the Paris hospitals, he inoculated various animals in different ways—as a result, obtaining a series of symptoms in the inoculated animals of such unvarying character that he classed them under the same head, and considered them to be due to the same cause. Taking the source of his original material into consideration, he felt justified in saying that what symptoms he obtained in his experiments were those of rabies, and that the material with which his inoculations were made contained the specific virus of that disease.

Going on with his work, and extending it in different directions, he announced, first, that by various modifications in treatment to which this "virus" could be subjected its strength could be altered and diminished, and that, by inoculating animals with a virus thus modified an immunity could be established in animals thus treated, against an inoculation with virus of the greatest strength, or against the bite of a rabid animal. Reversing this process, he found that the protective influence was equally established, if the protective inoculations were made *after* the injection of the strong virus, or the bite, had occurred, provided always that the period of incubation of the disease had not passed, and that the symptoms had not made their appearance.

These propositions he proved, or seemed to prove, by a very large number of experiments upon animals, and those most intimate with the results obtained were satisfied that healthy animals—dogs in particular—

could be protected by the measures which he suggested from an attack of rabies after being bitten by a rabid animal.

The next step in this work of Pasteur's, and the one which has attracted so much attention all over the world, was the application to human beings of his methods for the prevention of rabies after a bite by a rabid animal. The preventive inoculations against rabies in human beings began in the summer of 1885, and have been continued ever since, the statistics of results being furnished to the world by means of occasional summaries in the *Comptes Rendus.*

The criticisms to be made against Pasteur in connection with this work are, in the first place, that he fails to give as full details of the individual experiments by which his results have been obtained, as he should have done. By this omission, ground for dissent is given where none might have existed, if scientific men had had the full data before them. In the second place, this omission of detail makes it impossible to know—certainly from any published work of Pasteur's—whether his so-called "virus of rabies," with which his experiments of to-day are conducted, is the continuation of the virus originally obtained from one child, or whether he has taken the opportunity to control the original virus, by obtaining a fresh supply from rabid animals or men coming under observation since the time his investigations first began. In other words, do all of the results which he has obtained depend for their justification upon the reality of a case occurring a number of years ago?

The conditions of the case being as stated, it is hardly to be wondered at that the method of preventive inoculation against rabies has been subjected to severe animadversions, which have at times included its author, and, because the conditions are such as they are, independent observations, and control experiments by, as far as possible, unprejudiced observers are absolutely necessary before the full acceptance of Pasteur's assertions can fairly be demanded of the scientific world at large.

Such independent observations and control experiments may be made to cover a very wide field of investigation, but would include, first of all, the question as to whether others can obtain the same results as does Pasteur—using the same virus, in the same way that he does; secondly, whether this virus which he uses is the true virus of rabies,—a point which will be spoken of more at length further on; and lastly, a more successful attempt than has yet been made to discover the active principle in the material used for inoculation; this active principle seeming, by analogy, to be something with a power of self-reproduction.

So far as concerns the interests of humanity at large, the first is the most important point to be considered, and the endeavor must be made to find out whether—independently of Pasteur's laboratory—there is a virus which, when inoculated in animals, produces the results claimed; whether that virus can be modified in its strength by the treatment he proposes; and lastly, whether, by using this modified virus in a certain

way, an attack of rabies can be prevented. As a corollary to these, the question must be answered as far as possible whether any other material treated in the same way will produce the same symptoms when injected into animals.

It is evidence upon these points in particular, which it is desired to present in the following pages.

Upon the 2d of last July (1886), I received from Dr. Hamilton Osgood two rabbits, one of which had been inoculated upon the 19th or 20th of June, in Pasteur's laboratory, and had died on the 28th of June, and been kept upon ice since that time; the second of which was inoculated on the 21st of June in Pasteur's laboratory by Pasteur himself, and in Dr. Osgood's presence, and, alive when received, died on the night of July 4th. From these two rabbits the material for the series of experiments given in this paper was obtained, and the authenticity of the virus, as coming from Pasteur, is unquestionable.

The method of inoculation is well known, but a brief recapitulation will do no harm. The cord of a rabbit or other animal dead of rabies is removed as quickly as possible after death, and with all precautions against contamination. A small portion of this cord, of the medulla, or of the brain, is first beaten up in a little distilled or sterilized water to soften it, and then an emulsion is made of it by beating up this mixture with a small amount of neutral sterilized bouillon. This mixture is allowed to stand, and when the larger portions of the nervous tissue have sunk to the bottom of the vessel, the supernatant cloudy fluid is used as an injecting material. This entire process, against which much criticism has been directed, is merely *a method* by which a convenient form of injecting mass may be obtained. There is nothing in the least mysterious about it—water alone, bouillon alone, or any other equally bland and innocuous fluid may be employed, but in following an experimenter's work his exact methods should be used, and that has been done in this case.

Upon the receipt of the rabbits spoken of, the first question was, do the cords of these animals contain such material that, when inoculated into others, a constant series of symptoms will be produced with such unvarying regularity that the term "specific" can be applied to the cause producing them? With a view to determining this question, inoculations were begun at that time, and have been continued ever since, with the results given below. The inoculations given in this table were all made by trephining the skull, and the injection, immediately under the dura mater—not into the brain substance—of a very small amount of the fluid prepared as described above. At the head of the table stands the abbreviated record of the two rabbits inoculated in Pasteur's laboratory—including the date of inoculation and of death. Following this comes a tabulated statement of the rabbits which have been inocu-

lated in the regular way by trephining for the purpose of carrying on the virus and of observing the manner of symptoms resulting from such inoculations. The first column gives the number of the rabbits in this table; the second column (in Roman numerals) shows the number, as it stands in the original notes, of the animal from which the virus was taken; the third, fourth, and fifth columns explain themselves.

Table I.

First rabbit inoculated in Paris, June 19 or 20, 1886. Dead June 28th. Received July 2d.

Second rabbit inoculated in Paris, June 21st. Received July 2d. Died July 4th.

All of the inoculations given below were done by trephining.

No.	Inoculated from	Date of inoculation.	Appearance of symptoms.	Date of death.
1	I.	July 2, 1886.	July 14, 1886.	July 20, 1886.
2	I.	" 2, "	" 14, "	" 19, "
3	II.	" 5, "	" 28, "	August 2, "
4	II.	" 5, "	" 14, "	July 20, "
5	VI.	" 20, "	" 29, "	August 2, "
6	VI.	" 20, "	" 29, "	" 6, "
7	V.	August 25, "	Sept. 2, "	September 7, "
8	V.	" 25, "	" 2, "	" 12, "
9	XI.	Sept. 7, "	" 13, " (bites.)	" 20, "
			" 14, " (paralysis.)	
10	XI.	" 7, "	" 13, " (bites.)	October 2, "
			" 28, " (paralysis.)	
11	XIII.	" 22, "	" 29, "	" 3, "
12	XIII.	" 24, "	October 4, "	" 7, "
13	XVI.	October 4, "	" 11, "	" 16, "
14	Guinea-pig, II.	" 11, "	" 17, "	" 24, "
15	Guinea-pig, II.	" 11, "	" 14, "	" 22, "
16	XXI.	" 18, "	" 27, "	November 1, "
17	XXI.	" 18, "	" 25, "	October 30, "
18	XXI.	" 18, "	" 24, "	" 30, "
19	XXI.	" 18, "	" 27, "	November 1, "
20	XXIV.	" 22, "	" 30, " (very sluggish.)	" 8, "
			November 1, " (paralysis.)	
21	XXIII.	" 29, "	" 5, "	" 10, "
22	XXX.	Nov. 3, "	" 19, "	" 21, "
23	XXIX.	" 5, "	" 13, "	" 17, "
24	XLII.	" 17, "	" 24, "	December 1, "
25	Dog, V.	" 26, "	Decemb'r 3, "	" 10, "
26	Dog, V.	" 26, "	" 3, "	" 9, "
27	LXXIII.	Dec. 10, "	" 17, "	" 27, "
28	LXXIII.	" 10, "	" 17, "	" 24, "
29	LXXV.	" 24, "	" 30, "	January 3, 1887.
30	LXXVI.	" 24, "	" 31, "	" 6, "
31	LXXVIII.	January 8, 1887.	January 17, 1887.	" 22, "
32	LXXVIII.	" 8, "	" 16, "	" 22, "

Table II.

Statistics from the trephined rabbits in Table I.

No.	Time from the inoculation to the appearance of symptoms.	Time from the appearance of symptoms to death.	Time from the inoculation to death.
1	12 days.	6 days.	18 days.
2	12 "	5 "	17 "
3	23 "	5 "	28 "
4	9 "	6 "	15 "
5	9 "	4 "	13 "
6	9 "	8 "	17 "
7	8 "	5 "	13 "
8	8 "	10 "	18 "
9	6 "	7 "	13 "
10	6 "	19 "	25 "
11	7 "	4 "	11 "
12	10 "	3 "	13 "
13	7 "	5 "	12 "
14	6 "	7 "	13 "
15	3 "	8 "	11 "
16	9 "	5 "	14 "
17	7 "	5 "	12 "
18	6 "	6 "	12 "
19	9 "	5 "	14 "
20	8 "	9 "	17 "
21	7 "	5 "	12 "
22	16 "	2 "	18 "
23	8 "	4 "	12 "
24	7 "	7 "	14 "
25	7 "	7 "	14 "
26	11 "	6 "	17 "
27	7 "	10 "	17 "
28	7 "	7 "	14 "
29	6 "	4 "	10 "
30	7 "	7 "	14 "
31	9 "	5 "	14 "
32	8 "	6 "	14 "

Table III.

Averages, etc., from Table II.

Shortest period of incubation after trephining	3 days.
Longest " " " "	23 "
Average " " " "	8.6 "
Shortest period from the appearance of symptoms to death	2 "
Longest " " " " "	19 "
Average " " " " "	6.4 "
Shortest period from inoculation to death	10 "
Longest " " "	28 "
Average " " "	14.9 "

From these tables it may be seen, *first*, that, by inoculating animals —rabbits—in the way prescribed—*i. e.*, by trephining and injecting a small portion of an emulsion of a "rabid" cord under the meninges, certain symptoms occur after a period of incubation of varying duration, and that these symptoms are invariably followed by death. *Secondly*, that while the variation in length, of the period of incubation,

and of the time from the appearance of the symptoms until death, varies considerably between individual cases, nevertheless the *average duration* of each of these periods corresponds very closely indeed with the figures given by Pasteur, and quite as closely as a so comparatively small number of experiments could be expected to do.

This correspondence of results is very interesting and important—furnishing, as it does, the first firm ground of belief which would induce a sceptic to go on with further investigations.

The *symptoms* found in all of these animals have been practically identical, so that a description of one case furnishes a picture of what occurred in all. After the operation of trephining and inoculation was completed, the animal would remain stupid from the effects of ether for from fifteen minutes to half an hour, but sometimes not even so long. The most successful operations are performed with practically no loss of blood. After the effects of the ether have passed, the rabbit is apparently as well and bright as ever, and certainly, if one may judge by its appetite, it suffers no shock or any other disturbance from the operation.

Seen at the time and for some days afterward, there is not the slightest sign of any event having occurred out of its ordinary experience. The first thing abnormal which usually makes its appearance, or which is noticed, is a slight weakness of the hind legs—a slight unsteadiness as the animal jumps from one side of the cage to the other. This unsteadiness very often disappears entirely, if the animal be forced to make three or four jumps; it is as if a little stiffness or lameness were removed by exercise. This weakness sometimes makes its appearance suddenly, being very marked in animals which fifteen minutes before were seen to be perfectly strong, so far as close scrutiny could detect.

Before this beginning paralysis can be detected, there is, occasionally, a decided change in the character of the animal; from being lively and affectionate it becomes dull and sluggish, and even fierce—if such a term may be applied to a rabbit; in the latter case it will jump at and bite objects held toward it, and may even growl and spit at them, showing every evidence of a desire to do harm. Such a condition as this is not common, however, and the first symptom which makes a decided impression upon the observer is, usually, that of the unsteadiness of the hind legs already spoken of. This paralysis of the hind legs is progressive in intensity and in character. It passes from the posterior extremities to the anterior, and increases until, at last, the animal lies upon its side entirely unable to move. It is, distinctly, a loss of motor power; reflex action remains until life is actually extinct. The power of deglutition is lost in twenty-four or more hours from the first appearance of any symptoms—and it was at one time supposed that death was caused by starvation. This can hardly be the case, however, inasmuch as the stomach is always full of partially digested food, and the tissues are not

sufficiently wasted to warrant the supposition. Opisthotonos is early marked, and continues in an extreme degree after life is extinct. Before the paralysis has progressed so far that no motion is possible—there is at times a very considerable degree of restlessness—the animal struggles laboriously from one side of the cage to the other, without apparent object, falling over at almost every step. Just before death, there seems to be a revival of the powers—as manifested by a renewal of these struggles; —these movements disappear, absolute quiet ensues, and death occurs so easily that it is oftentimes impossible to tell whether life is or is not extinct. Whilst the symptoms last, the pulse, respiration, and temperature vary in an extreme degree, from very low to very high, and *vice versâ;* the variations being entirely without rhythm or symmetry, and apparently coinciding somewhat with the amount of muscular effort the animal may be making at the time. There is never any diarrhœa, no increase in salivary secretion, and no constant or peculiar change in the appearance of the mucus membrane of the mouth or fauces.

These are the appearances which have been presented in every animal recorded in this paper; not only in those which have been inoculated by trephining, but also in those which have succumbed to subcutaneous injection.

The appearances seen upon post-mortem examination are as uniform as are the symptoms which precede death; and, again, a description of one animal is practically a description of all. Naturally, the point of inoculation and the nervous tissues are the objects of greatest interest. If the inoculation has been by subcutaneous injection, the puncture through the skin may sometimes be made out by the presence of a minute spot of pigmentation, but there is never any sign of a local disturbance. If trephining has been performed, there is sometimes to be seen a very slight elevation of the brain substance under the point in the skull from which the button of bone has been removed. This is as often absent as present, but might be expected to occur by reason of the lack of the usual pressure at the point where the bone has been disturbed. There is never to be seen any sign of suppuration at the point of inoculation, or, for that matter, anywhere else; nor is there any other indication of a local disturbance. In *all cases*, however, whether the inoculation has been by trephining or by subcutaneous injection, the removal of the calvarium shows a congested condition of the membranes of the brain, without, however, any adhesive inflammation of the meninges. The cerebral sinuses are full of dark, clotted blood, but beyond this there is nothing to be seen in the brain. The brain substance is normal in color and consistency, and there are no spots indicating hemorrhage or emboli. The same conditions hold upon laying bare the spinal cord. The vessels of its membranes are sufficiently congested to give it a pinkish look, which disappears almost entirely when these membranes are removed,

and the cord itself presents to the eye almost precisely the glistening white appearance of that of an animal which has been killed by chloroform or ether. In this case, as in the brain, the color and consistency are normal. (It has seemed to me as if there was more congestion in the venous sinuses of the cord membranes in the sacral, lower dorsal, and cervical regions; whether this is accounted for by the anatomical structures of the parts, I am not sufficiently skilled in comparative anatomy to decide.)

The internal organs of the thorax and abdomen present nothing unusual. The heart is always full of dark blood, and has ceased beating in diastole. The lungs are healthy in appearance, of a bright red color, and sometimes show localized spots of a dark red, which are situated on the side upon which the animal was lying when it died. The stomach is usually full of half-digested food, and occasionally shows spots of post-mortem digestion, along the greater curvature for the most part. There is not any marked congestion of its mucous membrane. The small intestine is empty; the rectum full of well-formed normal feces; the bladder is sometimes enormously distended with a clear, amber-colored urine of a low specific gravity and alkaline reaction; at other times it contains a moderate amount of fluid, with a very heavy deposit. A sample analysis by Drs. Wood and Harrington, of the Harvard Medical School, is as follows: Sp. gr. 1.036; reaction very acid; sediment excessive; albumen 0; sediment: amorphous urates, acid urate of sodium, spermatozoa, and mucous casts, some of which were evidently from the kidneys.

The kidneys are normal in appearance; the liver and spleen are sometimes full of blood, and, rarely, somewhat anæmic; the vessels of the mesentery and of the skin are always full of blood, so that the smallest branches may be easily traced.

In none of the animals upon which post-mortem examinations have been made has there been noticed the extreme degree of congestion of the internal organs upon which so much stress has been laid. The absence of this appearance is at least in part readily explained. The animals here spoken of were, during the whole time they were under observation, kept isolated and free from any disturbance whatever, so that no excitement other than that incidental to the disease was produced. On the other hand, clinically speaking, the instant an animal is suspected of rabies it is persecuted and chased from one corner to another, until, unless frightened to death, it is put out of existence by clubs and stones. Under such conditions as these, congestion and even hemorrhage of the internal organs are but the natural result. The absence of the classic "period of excitement" in so many of the animals here recorded may very probably be ascribed to the same circumstances of quiet surroundings and freedom from annoyance. This "period of excitement" is all that makes the

difference in the books between the "furious" and the "dumb" forms of rabies. It precedes the appearance of paralysis, if it occurs at all, and is always terminated by the paralysis.

Taking into consideration the condition of things as it must be in the brain and cord, with each individual cell struggling against some foreign element, it is not difficult to understand how easily these disturbed cells may send various messages to the muscles, and how easily these messages may result in manifestations of excitement. From such a point of view, it is hardly rational to consider the "furious stage" as anything more than a chance appearance in the course of rabies, the likelihood of its presence being diminished as those things tending to excitement are taken away.

In order not to give any ground for the criticism that the results in this paper are too favorable, and in order not to claim too great perfection for the operator's skill, every animal used in this investigation will be spoken of, and where a variation from the results expected has occurred, the explanation, if there is any, will be given in accordance with the facts observed.

The first series of experiments which comes under this head is that which includes those rabbits which were meant to show the usual symptoms—were inoculated for that purpose—and yet failed to do so. These experiments will be found in the following table:

TABLE IV.

No.	Date and method of inoculation.	Result and explanation.
1	Inoculated by trephining, Sept. 22, 1886.	Dead, September 23. Blood-clot on brain. Injury to longitudinal sinus.
2	Inoculated by trephining, Sept. 24, 1886.	No symptoms. Killed Dec. 21, 1886. Dura mater not pierced by syringe needle, and virus washed out.
3	Inoculated by trephining, Oct. 4, 1886.	Savage Oct. 13 to 23, then no signs. Killed on December 21, 1886. Virus partially destroyed (attenuated) by heating.
4	Inoculated by trephining, Oct. 22, 1886.	Sluggish Oct. 30; savage Nov. 5; no signs on 7th. Killed Dec. 22. Virus partially destroyed by heating (attenuated).
5	Inoculated by trephining, Oct. 29, 1886.	Slight paresis Nov. 6; none on 7th; savage 10th to 14th. Killed Dec. 22, 1886. Virus attenuated by heating.
6	Inoculated by trephining, Nov. 3, 1886.	No signs. Killed December 23, 1886. Virus destroyed by heating.
7	Inoculated by trephining, Nov. 5, 1886.	Sluggish on 14th and 15th, then well. Killed December 23, 1886. Virus attenuated by heating.
8	Inoculated by trephining, Nov. 17, 1886.	No signs thus far, January 25, 1887. In this case syringe leaked, and it is doubtful if any of the material was introduced.

Thus it appears that there are eight animals which may be counted as failures; that is to say, the results were not as they were intended to be. Of three, the note appended to Nos. 1, 2, and 8, explains itself; of the others, Nos. 3 to 7, it is stated that the virus was more or less affected by heating in all of them, and this needs a little explanation. The way in which this mistake—for it was one in these cases—occurred, was as follows: It was not always possible to use the animals for inoculation immediately after death, and where this was the case, the bodies were at once frozen to prevent any change. In preparing some of these animals for use they were placed, by a mistaken idea of an assistant, in the *steam sterilizer* in order to hasten the thawing process. The heating was carried too far, and, as a result, the strength of the virus was diminished or destroyed entirely. This table, therefore, does not in the least weaken the force of the first one, but rather seems to show what may happen even when the operator himself takes all the precautions required. The cases where the heating of the animals occurred are also of value, as showing how readily a rise of temperature affects the virus in the cords.

This point will be found illustrated further on by experiments purposely made to test it.

Passing from obtaining such systematic results as are detailed in Table I., the questions arise, How does a variation of the inoculating material influence the result? Does a change in the fluid used produce a change in the symptoms, or are these symptoms simply the result of the introduction of any foreign substance under the meninges of the brain? This point, put in the form of the broad assertion, that nothing but this virus will produce these symptoms, would have to be supported by a very large series of experiments, and this has not been attempted. That variations, or rather a non-appearance of the symptoms, may be the result of changes in the material introduced, is well shown in the accompanying summary.

Table V.

Control experiments upon rabbits.

No.	Date of inoculation.	Method and material used.	Result.	
1	Aug. 2, 1886.	By trephining. Virus heated 5 m. to 110° F.	No symptoms.	Well, January 28, 1887.
2	Aug. 2, 1886.	The same.	No symptoms.	Well, January 28, 1887.
3	Sept. 24, 1886.	By trephining. Bouillon and water without any cord.	No symptoms.	Well, January 28, 1887.
4	Sept. 24, 1886.	The same.	No symptoms.	Well, January 28, 1887.

No.	Date of inoculation.	Method and material used.	Result.
5	Oct. 11, 1886.	By trephining. Emulsion of cord dried at 20° C. for ten days.	Very slight paresis, October 20th and 21st. Well, January 28, 1887.
6	Oct. 11, 1886.	The same.	Very slight paresis, October 21st. Well, January 28, 1887.
7	Oct. 19, 1886.	By trephining. Emulsion of cord of healthy rabbit.	No symptoms. Well, January 28, 1887.
8	Oct. 19, 1886.	The same.	Died *suddenly*, Nov. 19, 1886. Liver stuffed full of coccidium oviforme nodules.
9	Oct. 19, 1886.	The same.	Died *suddenly*, Nov. 7, 1886. Liver stuffed full of coccidium oviforme nodules.
10	Oct. 19, 1886.	The same.	No symptoms. Well, January 28, 1887.

The results here tabulated show that in every instance—other conditions being exactly the same—of inoculation by trephining with material different from the usual emulsion of the cord of an animal dead with the paralytic symptoms, nothing occurred which could by any possibility be ascribed to the inoculations except twice. The two cases where slight paresis was observed (5 and 6) only indicate that the strength of the virus was diminished by the drying of the cord used. The two cases in which death occurred (8 and 9) showed absolutely none of the regular signs; there was no change in disposition, no paralysis, no period of incubation, and the post-mortem examination showed the presence of enormous numbers of what looked like miliary tubercles with a few small abscesses. These changes were confined to the liver in both cases. Upon microscopic examination these nodules were found to be *not* produced by tuberculosis, but by the "coccidium oviforme"—a parasite not uncommon in rabbits. (Leuckart, *Parasites of Man*, 1886, p. 203, *et. seq.*)

These results, together with all the others mentioned in this paper, teach us that suppuration is not only not a necessary accompaniment of inoculations after Pasteur's method, but that the moment suppuration appears the experiment is vitiated, so far as proving or disproving anything of Pasteur's assertions is concerned. This is of special importance in connection with Spitzka's paper published in *The Journal of Comparative Medicine and Surgery* for July, 1886, under the title of "How Can We Prevent False Hydrophobia." This author distinctly states that he does not wish to touch upon the question of the existence of such a disease as true rabies, but, at the same time, his authority is quoted, as well as his experiments, to prove that there is no such thing, and, *especially*, that any putrescible substance will produce regularly, after a period of incubation, the paralytic symptoms claimed to arise

from a specific virus. Surely there is nothing in six experiments, incompletely reported, to warrant such an assertion. In the account of the autopsies, only two are given; *purulent meningitis* was found in one case, and *inflammatory red softening* of great extent in the other, together with, in this latter case, a blood-clot, and congestion and tumefaction of the hemisphere on the side upon which the operation was performed! Of course, if any results whatever are to be quoted as arguments in such a case as this, and if symptoms due to any sort of injury to the brain with results differing entirely from those of Pasteur are to be considered as disproving the occurrence of uniform results and pathological appearances, discussion is useless.

Still more striking than any of the results already detailed, were a series of experiments made upon guinea-pigs. These experiments were additional control work, to determine the *specific* nature of the virus employed, and the results obtained, in view of work done at other times, are exceedingly emphatic. An emulsion of the cord and medulla of a rabbit dead with the usual symptoms was prepared in the usual way, and three minims of the solution were injected subcutaneously in the right groin of the full-grown, healthy guinea-pigs. The usual precautions against contamination were taken, and the results are given in the following table.

TABLE VI.

Subcutaneous inoculation of guinea-pigs.

No.	Date of inoculation.	Date of appearance of symptoms.	Result.
1	Sept. 22, 1886.	Sluggish, and slight paresis, October 11th to 16th. Well on 17th.	No symptoms from October 17th. Killed December 27th. No changes.
2	Sept. 22, 1886.	Paresis October 9th.	Dead Oct. 10th. Usual congestion of membranes of cord.
3	Sept. 22, 1886.	No symptoms.	Killed Dec. 27th. No changes.
4	Sept. 22, 1886.	Paresis October 13th.	Dead Oct. 16th. Usual congestion of membranes of brain and cord.
5	Sept. 22, 1886.	No symptoms.	Killed Dec. 27th. No changes.
6	Sept. 22, 1886.	No symptoms.	Killed Dec. 27th. No changes.
7	Sept. 22, 1886.	No symptoms.	Killed Dec. 29th. No changes.
8	Sept. 22, 1886.	No symptoms.	Killed Dec. 29th. No changes.
9	Sept. 22, 1886.	Paresis October 9th.	Dead Oct. 3d. Usual congestion of membranes of brain and cord.
10	Sept. 22, 1886.	No symptoms.	Killed Dec. 29th. No changes.

Here it may be seen, that in four out of the ten animals inoculated symptoms made their appearance which could be referred only to a disturbance of the nervous centres, and in three of these four animals the appearance of the symptoms was followed by an increase in their severity

and extent with a fatal termination. This result is especially striking in view of the fact that in other experiments a very large number of inoculations have been made with all kinds of pathogenic and non-pathogenic bacteria, in the same way, and in the same species of animal, and yet the writer has never seen any result like that here recorded. Lesions extending from a miliary abscess at the point of inoculation to a general septicæmic or tuberculous process have been obtained, but never under any circumstances anything which seemed in the least like a disturbance of the nervous centres. To a practical bacteriologist, therefore, the results given in the preceding table are especially convincing. In no case was there the slightest indication of a local disturbance at the point of inoculation; the skin was as if it had never been touched, so much so, that recourse to the record was necessary to determine where the inoculation had been made.

As a further proof that the symptoms observed in the dead guinea-pigs were in reality due to the same cause as was producing similar conditions in rabbits all the time, the cord of one of the animals was removed, beaten up with distilled water and bouillon in the usual way, and inoculated by trephining into two rabbits. The result was confirmatory, the two animals showing the usual symptoms; the appearance of weakness of the hind legs increasing to general paralysis followed by death. These results are tabulated below.

TABLE VII.

Test of cord of guinea-pig upon rabbits.

No.	Date and method of inoculation.	Appearance of symptoms.	Result.
1	By trephining. Oct. 11, 1886.	Symptoms of paresis Dec. 12th.	Death Oct. 24th, with the usual appearances in membranes of brain and cord.
2	By trephining. Oct. 11, 1886.	Sluggish on 14th, paresis on 15th.	Death Oct. 22d, with the usual appearances in membranes of brain and cord.

Of course, no such investigation as the present would be complete unless observations were also made upon dogs. These animals were used, but not to anything like the same extent as rabbits, for the reason that the behavior of the "specific virus" in rabbits is the point upon which the greatest stress is laid and upon which so much depends. In the dogs experimented upon, the conditions were made precisely the same as obtained in the work upon rabbits. The instruments were sterilized with the same regularity, the operations were performed in the same way, and the material used for inoculation was, as in the other cases, an emulsion of the brain and cord of an animal previously inoculated with similar material. Another reason for not making more

numerous experiments upon dogs is the fact that in every case in which the conditions of a successful inoculation have been fulfilled, the result has been the same, the production of identically the same symptoms in dogs as have always appeared in rabbits treated in the same way; these symptoms coming on after a "period of incubation" extending over from eight to twelve days, then increasing in severity, and finally terminating in death.

The result of these experiments upon dogs is given in the accompanying table:

TABLE VIII.

Inoculation experiments upon dogs.

No.	Manner and date of inoculation.	Symptoms appeared.	Result.
1	By trephining. Nov. 9, 1886.	Paresis, Nov. 21st.	Dead, Nov. 25, 1886.
2	By trephining. Dec. 10, 1886.	Paresis, Dec. 20th.	Dead, Dec. 26, 1886.
3	By trephining. Dec. 10, 1886.	Paresis, Dec. 20th.	Dead, Dec. 26, 1886.
4	By trephining. Dec. 15, 1886.	Paresis, Dec. 20th.	Dead, Dec. 27, 1886.
5	By trephining. Dec. 15, 1886.	Dilated pupils, Dec. 24th; paresis, Dec. 26th.	Dead, Dec. 30, 1886.

In order to be sure of the virus, the cord of one of these dogs was tested by inoculating into rabbits, with the production of the usual symptoms; the rabbits were trephined and inoculated in the usual way; the paralysis made its appearance at the usual time, spread in the usual way, and ended fatally, as usual. The results are given in Table IX.

TABLE IX.

Test of cord of dogs upon rabbits.

No.	Date and method of inoculation.	Symptoms appeared.	Result.
1	Trephined, Nov. 26th.	Symptoms. Dec. 3d.	Death, Dec. 10th.
2	Trephined, Nov. 26th.	Symptoms. Dec. 3d.	Death, Dec. 9th.

This is but a small number of experiments, taken by themselves; their exact correspondence with those made upon other animals is their essential value. In every case, not only were the symptoms during life the same as those observed in rabbits and guinea-pigs, but the appearance upon post-mortem examination was the same, with the single exception that the stomach was entirely empty, except in two cases, in which a small amount of *hair* was found. (In rabbits the stomachs were always full, although this cannot be said to mean much after all, in the face of the assertion of physiologists that one can never empty a

rabbit's stomach, even by prolonged starvation.) In none of the dogs were there any prolonged periods of excitement or fury seen; such a condition as the "furious" stage was entirely absent. They were all subject to short fits of restlessness, during which they moved quickly and aimlessly from one side of the cage to the other. At these times they would occasionally cry out, and sometimes would appear as if in extreme pain; shortly afterward they would sink into a sort of lethargic state, from which it was extremely difficult to rouse them. To what extent these short periods of restlessness would have been extended if the animals had been at large, and how near they would have approached the classic "fury" of a rabid dog, it is, of course, impossible to say. As a matter of fact, the dogs were kept confined and entirely protected from any but the most necessary disturbance, and any alienist has daily experience of the sedative effect of quiet and seclusion upon his patients. In only one case was there any marked desire to bite, and that was only manifested after the animal had been disturbed, as would naturally be the case at any time with a dog sick of any disease. The membranes of the brain and cord were always congested, but there were no localized symptoms at the point of inoculation, nor any greater disturbance on the side where the inoculation had been made; most important of all, there was not the slightest indication of any suppuration.

Before concluding this part of the paper, which has had for its object the determination of the existence of the specific virus called that of rabies, the same thing is to be done here as in the case of the rabbits—the unexpected results and failures in the inoculation of dogs must be recorded. This is done in Table X.

TABLE X.

Failures of inoculations upon dogs.

No.	Date and method of inoculation.	Symptoms appeared.	Result.
1	Subcutaneous injection, Sept. 7, 1886.	No symptoms.	Killed Dec. 10th. No changes.
2	Subcutaneous injection, Sept. 7, 1886.	No symptoms.	Killed Dec. 10th. No changes.
3	Trephined, Oct. 29, 1886.	Symptoms, Nov. 4, 1886.	Dead Nov. 11th. Compression of brain by blood-clot.
4	Trephined, Dec. 10, 1886.	Symptoms, Dec. 11, 1886.	Dead Dec. 13th. Brain injured by trephine; spot of softening about point of injury.

The first two of these animals were inoculated by subcutaneous injection; after being kept under observation for three months they were killed, as no symptoms had appeared. The last two cases explain themselves: in the first, the longitudinal sinus was pierced, and in the second

the trephine slipped during the operation and entered the brain, the result being the same as would occur in any other such injury.

The work already recorded seems to show pretty definitely, that there is a specific virus contained in the cords of rabbits inoculated with the material and in the way spoken of by Pasteur. This point having been settled, the question arises, Is it true that by certain procedures this virus can be diminished in strength, and can animals be protected from an attack of rabies after inoculation with that disease by the method suggested by Pasteur—in other words, does "protection" protect? The remainder of the work done in this investigation has been completed with the object of illustrating this point.

The principle of "protective inoculation" is drawn from these facts: That the cord of an animal dead of rabies, if promptly removed and kept at an even temperature (20° C.), and in a dry atmosphere, will gradually lose its virulence, so that, at the end of ten or twelve days, this virulence will be entirely gone; and that this loss of virulence is gradual and progressive, *not* sudden. The *theory* is, that by injections on successive days, of emulsions of cords of increasing virulence, an immunity is established against an attack of the real disease, or, what is the same thing, against an inoculation with the strongest virus, and that this "immunity" is established whether the inoculation, or bite, occurs before, or after, the protective measures are taken. As a corollary, it follows, of course, that if the inoculation, or bite, occurs before protection, the protection, to be effective, must be completed before the period of incubation of the disease has passed; that is to say, before any deep impression has been made upon the nervous centres.

For the purpose of protective inoculation, cords are to be removed immediately after death with all possible precautions against contamination. They are then suspended in sterilized jars, stoppered with cotton-wool, and containing some hygroscopic material to promote the absorption of moisture. The jars are kept in a dry chamber, as nearly as possible at a temperature of 20° C., and portions of the cords they contain are used for injection as occasion requires,—the cords being made into an emulsion in the usual way. This is the method; but to have everything ready for a protective inoculation at any given time, some calculation is necessary, and a number of rabbits must have been inoculated, so that they will die on the day that it is necessary to begin drying the cords, so that these cords shall be of the proper intensity of virus on the days they are needed. This, for a single series of protective inoculations, when it is known at what date the protection must begin. If the desire is to be ready to perform these protective inoculations at any time, a little reflection will show that rabbits must be inoculated every day, at least one must die every day, and that the cord must be removed and

the drying begun every day. Of course, such work as this can be carried out only at the expense of much time, labor, and money, and it appears at once how much more easy it is to condemn the method by the facility with which inaccurate results may be obtained, than it is to test its merits thoroughly by exact observation.

In the work detailed below, the first procedure was preparing the rabbits so that the inoculations with the strong virus could be made upon the 10th of November, 1886, and the protective inoculations could be begun upon the following day. What was done may be thus tabulated: Rabbits had been previously inoculated so that it was possible on

TABLE XI.

Nov. 1. To prepare a cord for use on 11th (10 days).
" 3. " " " 12th (9 days).
" 5. " " " 13th (8 days).
" 7. " " " 14th (7 days).
" 9. " " " 15th (6 days).
" 10. Rabbits were inoculated with virus of full strength and protection begun.
" 11. With cord of 1st (10 days drying) and cord prepared for use on 16th
" 12. " 3d (9 ") [5 days.
" 13. " 5th (8 ") and cord prepared for use on 17th
" 14. " 7th (7 ") [4 days.
" 15. " 9th (6 ") and cord prepared for use on 18th
" 16. " 11th (5 ") [3 days.
" 17. " 13th (4 ") and cord prepared for use on 19th
" 18. " 15th (3 ") [2 days.
" 19. " 17th (2 ") and cord prepared for use on 20th [1 day.
" 20. " 19th (1 ") and protective inoculation was completed upon this day, the 20th.

The value of this method of procedure was tested upon four series of rabbits in four different ways: *First*, by the subcutaneous injection of virus of the strongest quality, and the following of this on successive days for ten days by the subcutaneous injection of virus of progressively increasing strength, as shown in the preceding table; *second*, by carrying out the protective measures, as above, first, and then injecting the strong virus; *third*, by inoculation with the strong virus first, *by trephining*, and then protecting by subcutaneous injection on ten successive days; *fourth*, by protection first by subcutaneous injection, and then inoculation with the strong virus *by trephining*.

The first test is unquestionably the most important and its results are especially striking, for the reasons that the conditions observed approach nearest to what occurs in real life, and because it is against such subcutaneous inoculation—by bites—that the protective measures are adopted in human beings. The results of the test in regard to the first point are given in the following table:

*

Table XII.

Protective inoculations in rabbits—1. By subcutaneous injection; inoculation first, and then protection.

No.	Date and method of inoculation.	Protective injections.	Result.
1	Nov. 10, 1886. Subcutaneous injection.	Protected Nov. 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, with cord 10, 9, 8, 7, 6, 5, 4, 3, 2, and 1 day old respectively.	No symptoms. Well Jan. 30, 1887.
2	The same.	The same.	The same.
3	The same.	The same.	The same.
4	The same.	The same.	The same.
5	The same.	The same.	The same.
6	The same.	The same.	The same.
7	The same.	The same.	The same.
8	The same.	The same.	The same.
9	The same.	The same.	The same.
		Symptoms Nov. 24.	Dead Nov. 28.

At the same time that these animals were inoculated, a second series was treated in the same way, and with the same virus, and left unprotected, as seen below.

Table XIII.

Control of virus used in Table XII. Subcutaneous injection of rabbits, without subsequent protection.

No.	Date and method of inoculation.	Symptoms.	Result.
1	Nov. 10, 1886. Subcutaneous injection.	No symptoms.	Well Jan. 30, 1887.
2	The same.	Symptoms Jan. 8, 1887, paresis.	Dead Jan. 12, 1887.
3	The same.	Symptoms Nov. 23, paresis.	Dead Nov. 27, 1886.
4	The same.	Symptoms: Fierce on Nov. 22; paresis on Nov. 27, 1886.	Dead Nov. 30, 1886.
5	The same.	Symptoms: Fierce on Nov. 28 to Dec. 8; paresis Dec. 8.	Dead Dec. 13, 1886.
6	The same.	Symptoms of paresis Nov. 27.	Dead Nov. 30, 1886.
7	The same.	Symptoms of paresis Dec. 3.	Dead Dec. 8, 1886.
8	The same.	No symptoms.	Well Jan. 30, 1887.
9	The same.	Symptoms of paresis Dec. 3.	Dead Dec. 7, 1886.

These two tables present a very strong contrast: upon the first, one death is recorded; and upon the second seven are noted. In the first case, the symptoms appeared on the fourth day after the protective injections were completed, and it is perfectly justifiable to suppose that in this case the period of incubation of the original inoculation was too short to permit the protective measures adopted to take full effect. On the other hand, the seven deaths recorded on Table XIII., out of nine rabbits, inoculated and left unprotected, show that the virus used was possessed of great active properties, and the inference is warrantable that some influence for good was exerted by the measures adopted in the first series, subsequent to the injection of the strong virus. The

symptoms of the rabbits which were affected were the same in all cases, and these and the gross pathological appearances correspond exactly with those already detailed, as seen in the rabbits in Table I.

The results of testing the protective injections in the three other ways spoken of, are given below in Tables XIV., XV., XVI., and XVII.

Table XIV.

Protective inoculation in rabbits—2. By subcutaneous injection; protection first, and inoculation with strong virus.

No.	Protected.	Inoculated.	Result.
1	Nov. 11 to 20, with cords 10 to 1 day old.	Subcutaneous injection with strong virus, Nov. 29, 1886.	No symptoms. Well Jan. 30, 1887.
2	The same.	The same.	The same.

Table XV.

Protective inoculations in rabbits—3. Inoculation by trephining; inoculation first, and then protection.

No.	Date and method of inoculation.	Protected.	Symptoms.	Result
1	Nov. 10, 1886. By trephining.	Protected Nov. 11 to 20, with cords 10 to 1 day old.	Paresis appeared Nov. 20.	Dead Nov 25.
2	The same.	The same.	Paresis appeared Nov. 21st.	Dead Nov. 25.
3	The same.	The same.	None.	Well Jan. 30, 1887.
4	The same.	The same.	Sluggish from Nov. 21 to 24, then well.	Well Jan. 30, 1887.

Table XVI.

Protective inoculations in rabbits—4. By trephining; protection first, then inoculation with strong virus by trephining.

No.	Protected.	Inoculated.	Result.
1	Nov. 11 to 20 with cord 10 to 1 day old.	Nov. 29 with strong virus, by trephining.	Sluggish Dec. 8; fierce to Jan. 1, 1887; well Jan. 30, 1887.
2	The same.	The same.	Paresis Dec. 7; dead Dec. 11.

Table XVII.

Attempted control of strength of virus used in Tables XV. and XVI.

Rabbits.

o.	Inoculated.	Result.
1	By trephining. Nov. 29, 1886.	No symptoms. Well January 30, 1887. (Syringe leaked badly during operation, and considerable hemorrhage occurred; probably no virus injected.)
2	The same.	Well January 30, 1887. (Longitudinal sinus opened during operation; enormous hemorrhage, and virus probably washed out.)

A study of these tables shows that no symptoms whatever occurred when the protection preceded the inoculation of the strong virus (Table

XIV.), this inoculation being made by subcutaneous injection nine days after the protective measures were completed. In Tables XV. and XVI., where the inoculation with the strong virus was made by trephining, there is recorded a loss of half the animals, when the protection preceded the inoculation, as well as when it followed it. These results are not as fatal as those of Frisch, of Vienna (*Wien. med. Presse*, Nos. 31 and 32, 1886), and, in any case, the objection that this is an unjustly severe test of the method is a valid one, the inoculations by trephining bringing the virus in immediate contact with its place of election, and not allowing for the longer period of incubation which follows a subcutaneous inoculation (bite), against which method of inoculation the preventive injections are directed.

The last table (XVII.) gives the result of two inoculations by trephining, made to test the strength of the material used in the experiments recorded in Tables XV. and XVI. They were both failures—the result being undoubtedly due to the imperfect operation, which latter was caused by haste, owing to pressure of other work. That the virus was of the full strength is shown by the results in Tables XV. and XVI., and also by the symptoms in the animal from which it was taken, and in other animals inoculated with it to carry on the virus.

The work above recorded is what has been done to prove the three propositions:

(1) Is there a specific virus in the brains and cords of rabbits inoculated with Pasteur's material and after his methods?

(2) Does the treatment by drying, proposed by him, modify the strength of this virus? And, finally,

(3) Does injection with such "modified virus" produce an immunity against an inoculation (or bite) with virus of full strength?

The experiments have been given in detail, and, with the utmost frankness, every animal experimented upon has been spoken of, and absolutely nothing has been concealed. The writer began the investigation as a sceptic—as a disbeliever in the existence of a virus which would produce such peculiar symptoms as were claimed; he had not visited Pasteur, was ignorant of his precise methods, and knew nothing more of his results than he has seen fit to publish.

The results of the work here given must speak for themselves: they certainly seem to justify a change from scepticism, complete and absolute, to a belief in, at least, a portion of Pasteur's assertions. Whether the virus used in these experiments is the virus of true rabies or not is a point which cannot be settled until an opportunity is offered for investigation of the result of inoculations with material from a fresh case of rabies in a dog or other animal—*unless* such evidence has already been obtained in Pasteur's laboratory, and *published*. If the latter is the

case such publication has escaped notice up to the date of finishing this paper.

Whether this is the true virus of rabies or not, it is something specific. It may be the true virus, or it may be the true virus altered in some degree by its long transmission from animal to animal, or it may be something entirely different and apart from the true virus; this is a point of mere scientific interest. The practical result is the same in either case, so far as concerns the application of the method to man. It is absurd to suppose that in a country like France, where rabies is so comparatively frequent, and in a series of cases so large and closely observed as that of Pasteur, a certain percentage, and that a large one, of the patients submitted to him, have not been bitten by rabid animals. This being true, even with the few deaths claimed and heralded as "failures," the percentage of fatal results among his cases is unquestionably much smaller than among those in which his method was not adopted.

Every one knows that now, in severe bites or lacerated wounds, more frequent, stronger, and repeated preventive injections are used than was the case at first. This change does not alter the *principle* of the method, however, and as it was that it was desired to test, the original *daily interval* injections were adopted in this research.

A discussion of the minute changes observed has been avoided. The study of the microscopical appearances of the various organs, especially of the brain and cord, together with the results of bacteriological research, form a part of the work by themselves. It is hoped that it may be possible to find something *specific* in this direction at some future time.

The conclusions which may be drawn from the work here recorded seem to be as follows:

I. There exists in the cords and brains of animals inoculated in Pasteur's laboratory a *specific virus*, capable of the production of similar symptoms through a long series of animals.

II. That these symptoms are produced with absolute certainty when the method of inoculation is by trephining the skull, and injection under the dura mater,—with less certainty when the inoculation is by subcutaneous injection.

III. That the strength of this virus is lessened when the cords containing it are removed from the animals and placed in a dry atmosphere at an even temperature.

IV. That the symptoms produced by the inoculation of this virus only appear after a certain period of incubation, distinctly shorter when the inoculation has been done by trephining, than when done by subcutaneous injection.

V. That injections of the virus, modified in strength by drying, and

in the manner prescribed by Pasteur, exert a very marked protective influence against an inoculation with virus of full strength.

VI. That a very moderate degree of heat destroys the power of the virus entirely, whilst prolonged freezing does not injure it.

As will be seen, all of these conclusions are in complete accord with the declarations of Pasteur; their importance lies in the fact that they were reached at a distance from him, and by work entirely separated from any personal influence or bias.

The liberality of the Medical Faculty of Harvard University gave the necessary pecuniary support to this investigation. The energetic and persistent assistance of Mr. Austin Peters, M.R.C.V.S., permitted the completion of much of the work contained in it. Dr. Osgood's great trouble in obtaining and bringing the rabbits to this country, also deserves the author's most sincere thanks.

NOTE.—This paper was prepared for the press when the later results of von Frisch came under notice. The time to investigate the full paper was too short, and the summary as given in the *Medical Record* of January 22, 1887, was made use of. The conclusions to which he has come are precisely the same as those drawn from the work in this paper, so far as they cover the same ground. His further conclusions are based upon work done with virus obtained from street rabies, and seem to establish the identity between this and the experimental virus of Pasteur. His work has led him to the expression of a suspicion, already expressed by the writer in another place, that the intensive method of preventive treatment recently recommended by Pasteur, may very possibly produce the disease. Whether von Frisch is justified in his conclusions by a sufficient number of experiments upon which to base them, the writer cannot say at present; certainly the conclusions of his previous papers upon this subject were entirely too weighty for the foundation upon which they were built.

BOSTON, January 30, 1887.